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CONTENTS OF PREVIOUS NUMBERS

SEPTEMBER, 1919. NUMBER 3.

Leukocyte Counts During Influenza in Infants and Children. J. C. Montgomery, M.D., and E. C. Dunham, M.D., Baltimore.

Fat Metabolism of Infants and Young Children. IV. The Digestion of Some Vegetable Fats by Children on a Mixed Diet. L. Emmett Holt, M.D.; Angelia M. Courtney and Helen L. Fales, New York.

Cerebral Involvement in Hereditary Syphilis. Philip C. Jeans, M.D., St. Louis.

The Incidence and Significance of the Rheumatic Nodules in Children. Joseph Brennemann, M.D., Chicago.

Cerebral Complications in Mumps. Horton R. Casparis, M.D., Baltimore.

A Case of Diabetes Insipidus. David Murray Cowie, M.D., Ann Arbor, Mich.

Observations on the Toxic Properties of Heated and Decomposed Milk and of Milk Cultures of Bacterium Welchii (Bacillus Aerogenes Capsulatus). William W. Ford, M.D., Baltimore.

Progress in Pediatrics:
Review of Recent Literature on the New-Born. N. O. Pearce, M.D., Minneapolis.

Current Pediatric Literature.

OCTOBER, 1919. NUMBER 4.

The Caloric Requirements of Normal Infants and Children from Birth to Puberty. Fritz B. Talbot, M.D., Boston.

Influenzal Croup. Henry Lowndes Lynah, M.D., New York.

A Health Study of a Boy's School. Richard M. Smith, M.D., Boston.

Certain Nutritional Disorders of Children Associated with a Putrefactive Intestinal Flora. Langley Porter, M.D.; C. B. Morris, M.D., and K. F. Meyer, M.D., San Francisco.

Study of the Stools in Children's Institutions Showing the Incidence of Intestinal Parasitic Infections. L. R. DeBuys, B.S., M.D., New Orleans, and H. L. Dwyer, M.D., Kansas City, Mo.

Progress in Pediatrics: Review of Recent Literature on the New-Born. N. O. Pearce, M.D., Minneapolis.

Child Welfare. Report from International Congress.

Current Pediatric Literature.

NOVEMBER, 1919. NUMBER 5.

The Results of Thymus Extirpation in the Dog with a Review of the Experimental Literature on Thymus Extirpation. Edwards A. Park, M.D., and Roy D. McClure, M.D., Baltimore.

Current Pediatric Literature.

DECEMBER, 1919. NUMBER 6.

The Early Recognition of Hydrocephalus in Meningitis. Kenneth D. Blackfan, Baltimore.

Chemical Examination of the Blood in Children. A Study of One Hundred and Forty-Nine Cases. Henry Dwight Chapin, M.D., and Victor Carl Myers, Ph.D., New York.

The Rôle of the Antineuritic Vitamin in the Artificial Feeding of Infants. Amy L. Daniels, Ph.D., and Albert H. Byfield, M.D., with the Cooperation of Rosemary Loughlin, M.S., Iowa City, Iowa.

Stammering as a Disorder of Speech Dependent on Conditions of Child Development. Elmer L. Kenyon, M.D., Chicago.

Qualitative and Quantitative Changes in the Cerebrospinal Fluid of Various Diseases and Their Significance. A. Levinson, Chicago.

A Résumé of Some Experimental Studies on Cutaneous Hypersensitiveness. E. C. Fleischner, K. F. Meyer and E. B. Shaw, San Francisco.

Current Medical Literature.

Index to Volume 18.

JANUARY, 1920. NUMBER 1.

A Study of Achondroplasia, Introducing a New Symptom — Wedge-Shaped Vertebra. Thomas Foster Wheeldon, A.M., M.D., Richmond, Va.

The Food Value of the Milk of the Water Buffalo. William W. Cadbury, M.D., Canton, China.

Dextrose Tolerance in Atrophic Infants. P. M. Mattill, K. M. Mayer and L. W. Sauer, Chicago.

A Case of Hypersensitiveness to Cow's Milk. Edwards A. Park, M.D., Baltimore.

Clinical Department: A Case of Anomaly of the Diaphragm with Herniation into the Thorax of Certain Viscera Resulting in a Gastric and Intestinal Obstruction. L. R. DeBuys, B.S., M.D, New Orleans.

Progress in Pediatrics: Résumé on the Circulatory System. Review of Literature of 1917, 1918 and 1919. Henrietta Calhoun, M.B., Iowa City, Iowa.

FEBRUARY, 1920. NUMBER 2.

The Etiology of Arthritis Deformans in Children. Albert H. Byfield, M.D., Iowa City.

Calcium Metabolism of Infants and Young Children, and the Relation of Calcium to Fat Excretion in the Stools. L. Emmett Holt, M.D., Angelia M. Courtney and Helen L. Fales, New York.

Incidence of Protein Sensitization in the Normal Child. Horace Mitchell Baker, M.D., Boston.

Foreign Bodies in the Air and Food Passages. Edwin E. Graham, M.D., Philadelphia.

Auriculoventricular Heart Block in Children, with Report of a Case. J. A. E. Eyster and W. S. Middleton, Madison, Wis.

Abscess of the Lungs in Infants and Children. Harry Wessler, M.D., and Herman Schwarz, M.D., New York.

The Elimination of Acetone Bodies During Infectious Fevers. Borden S. Veeder, M.D., St. Louis, and Meredith R. Johnston, M.D., Captain, M. C., U. S. Army.

Clinical Department:
A Report of Two Cases of Congenital Stricture of the Esophagus. John Lovett Morse, A.M., M.D., Boston.

Progress in Pediatrics:
Recent Work in Anatomy, Physiology and Pathology of Infancy and Childhood. James B. Holmes, M.D., Baltimore.

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DILATATION OF THE COLON IN CHILDREN *

ALFRED EDWARD MEYERS, M.D.

SAN FRANCISCO

In reviewing the literature that has appeared on the subject of dilatation of the colon in children one is struck by the lack of definiteness pertaining to this condition. The various theories put forth endeavoring to point out the etiology of this abnormality are but a frank admission of our uncertainty. Likewise, the medical treatment of intestinal dilatation with its accompanying constipation is highly unsatisfactory, both to the parents of the patient and to the physician.

In the first place, What degree of hypertrophy and dilatation constitutes the so-called Hirschsprung's disease, otherwise known as congenital idiopathic dilatation of the colon, or megacolon? If one makes roentgenograms in all cases of persistent constipation, especially those beginning in infancy, he would find that many of the sigmoids are looped, twisted, redundant, dilated and hypertrophied. The past histories of these cases are practically identical. In spite of rational feeding during the early part of infancy, a bowel movement without the aid of a suppository, enema or laxative was a rare occurrence. Distention with gas was a noticeable feature, always causing abdominal protuberance. Normal growth was usually interfered with, and the patients were always below par. All seemed to fall an easy prey to the infectious diseases, thus manifesting an evident lowering of their resistance. But constipation was the dominant symptom which compelled them to seek medical advice. Lubricants such as paraffin oil, laxatives such as milk of magnesia or phenolphthalein, cathartics such as castor oil, and enemas of salt solution and oil were given daily with the hope that the bowel would sooner or later function normally, but the relief was only temporary, and the final result very unsatisfactory.

Correcting the diet received but very little attention, a factor very essential in combating the gaseous distention accompanying this condition. In 80 per cent. of the cases of chronic intestinal indigestion

* From the Department of Pediatrics, University of California Medical School.

one will find, on analyzing the diet, that the carbohydrates greatly predominate. The following diet is typical of that received by these patients:

Breakfast: Cereal, milk, toast, sometimes an egg.
Dinner: Meat (occasionally), vegetables (of all sorts), potatoes (in nearly every dietary), rice, spaghetti or macaroni. Desserts: puddings (tapioca or corn starch).
Supper: Soup, bread, milk or tea, cooked fruit or cake.

A glance will show that this dietary is unbalanced, and that the starchy elements make up nearly 80 per cent. of the food.

Regularity of habit is frequently tried, but the mother soon exhausts her patience in endeavoring to make the child try to move its bowels without the aid of some external stimulus, and the only encouragement she receives is that as the child grows older the intestines will take on their normal function. It is quite true that in a certain percentage of these cases, their little bodies grow up to meet this abnormality, but, as in many other conditions, Nature exacts her usual penalty.

Holt [1] gives the following as his interpretation of Hirschsprung's disease, or megacolon: "It is characterized by a great increase in the diameter of the colon and in the thickness of its wall. It was originally believed to be an idiopathic condition for which no sufficient anatomic cause could be found. Hence, it has been known as congenital or idiopathic dilatation of the colon." This seems to coincide with the views of most observers. From the same source the following definition of hypertrophic stenosis of the pylorus, with reference to its pathology, is taken: "The pathology of stenosis of the pylorus in early infancy is somewhat obscure and quite diverse views are held. It is believed by some that the primary and essential condition is one of spasm; that the hypertrophy, when it is present, is secondary; and that in many of the cases there is pylorospasm without hypertrophy.

The other view, and that which seems to harmonize best with the clinical symptoms and the pathologic findings, is that the primary condition is one of hypertrophy which is congenital; that spasm is added to this; that in all cases both factors — hypertrophy and spasm — are present; that while the cases differ in degree, they are the same in kind. Spasm certainly plays an important part in the production of symptoms; but to regard this condition as one essentially of muscular spasm seems to us erroneous." Thus we see that in the former condition (megacolon) there is hypertrophy (colon) and dilatation (sigmoid), and in the latter (pyloric stenosis) hypertrophy (pylorus) and dilata-

1. Holt, L. Emmett, and Howland, John: Diseases of Infancy and Childhood, Ed. 7, New York, D. Appleton & Co., 1918, pp. 321 and 408.

tion (stomach). Histologically, in congenital dilatation of the colon, the mucosa is twice its normal depth, contains pigment masses (chronic inflammation), while the muscularis mucosa is hypertrophied, especially in the region of the circular fibers. The microscopic picture of pyloric stenosis is very similar in that the circular muscle fibers are hypertrophied.

Enemas are essential in the treatment of megacolon, and attention is given to stimulating peristalsis by laxatives and cathartics as well as to correcting the diet. In hypertrophy of the pylorus, the dilated portion (stomach) is subjected to lavage, careful attention is given to the diet, and in many cases, especially where spasm is present, atropin is given with very good results. Dilatation of the colon does harm by predisposing to malnutrition, intestinal disturbances and intercurrent infections, while pyloric stenosis blocks the passage of the food into the system. Both conditions are treated medically and surgically with equally good results. The following table lists the symptoms as they appear in both conditions:

	Dilatation of the Colon	Pyloric Stenosis
Hypertrophy	Present	Present
Hypertrophy of circular muscle	"	"
Dilatation	"	"
Visible peristalsis	"	"
Constipation	"	"
Congenital malformation	"	"
Gastro-intestinal retention	"	"
Spasm	?	"

If, as has been shown, there is a similarity between megacolon and hypertrophic stenosis of the pylorus, and if, as is well known, there are different degrees of the latter, varying all the way from an occasional slight spasm, manifested by the vomiting of a few cubic centimeters of food, to the very severe type in which there is hypertrophy and spasm combined and wherein it is impossible for the child to retain even boiled water in the stomach, why is it not possible for all the dilated, elongated and redundant colons which we see in constipated children to constitute varying degrees of megacolon? It seems to me that it would help to clarify an obscure point in a condition not definitely understood if we could eliminate the term "Hirschsprung's disease" and classify all cases according to the amount of dilatation, slight, moderate, or great, as the case might be, the latter to embrace what is known as megacolon, and the former all those conditions showing from a slight to a moderate amount of redundancy.

In the second place, What is the true cause or exciting factors of dilatation of the colon? Perhaps the best classification of the various theories advanced is that given by Barrington-Ward:[2] (1) Mechanical; (2) neuropathic; (3) dilatation following colitis, and (4) congenital anomaly.

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2. Barrington-Ward, L. E.: Brit. J. Surg. **1**:345, 1914.

Many cases of dilated colons have been reported wherein the cause of the trouble was traced to a mechanical obstruction, but the fact that this condition has been reported in a premature child, that postmortems show no obstruction in many cases, and that constipation begins in the first weeks of infancy, seems sufficient proof against the theory of mechanical obstruction.

Walker and Griffith [3] advanced the idea that dilatation of the colon might be due to distention by gas following an attack by colitis. The same arguments as against the mechanical idea may be applied to this theory. It is true that the mucosa in megacolon shows evidence of chronic inflammation and very often the feces are covered with mucus, but both may be the result instead of the cause. It is easily conceivable that an unrecognized case of dilatation of the colon of a slight degree may be complicated with a colitis, which in turn favors the transition to one of greater degree. It is also possible that a colitis may be caused by fermentation usually present in a dilated colon.

The concensus of opinion seems to be that dilatation of the colon is a congenital abnormality in the lower end of the hind gut, which takes the form of a muscular hyperplasia. It usually involves the rectum and spreads a varying distance up the colon. The abnormality may affect one or all segments of the intestines. Thickening of the walls and increase in the lumen are caused by the hyperplasia of the muscle fibers. Considerable clinical and pathological evidence has been presented since Hirschsprung [4] gave his classical description supporting this view, but my experience with a case of megacolon and five other cases of partial dilatation, diagnosed from the history, clinical symptoms and roentgenogram of all the colons, leads me to believe that there is still another element concerned in this condition, that of spasm.

REPORT OF CASES

Case 1 (49630).—Mother and father are living and well. One brother, aged 7 years, is living and well. No children dead. No miscarriages. Mother's uncle was insane. No history of cancer, rheumatism or nervous diseases. Parents are receiving no treatment for any condition.

Past History.—Child was born in Nova Scotia. Delivery normal. Birth weight 7 pounds. Breast fed for nine months, when child was weaned and fed on Nestlé's food, cow's milk and pap. Present diet consists of cereal, turnips and other vegetables, potato and fruit. Meat was rarely given. With the exception of the present condition, patient has always been well. No operations or accidents. Sat up when 6 months of age; first teeth at 4 months; walked at 13 months; talked at 15 months. Always subject to croup.

Present Illness.—Constipation began as soon as the child was put on cow's milk and other food. Bowels moved regularly while on breast milk. Would go four days without defecation, if permitted, though mother usually gave an

3. Walker and Griffiths: Brit. M. J. **2**:230, 1893.
4. Hirschsprung: Jahrb. f. Kinderh. **32**:1, 1888.

enema at the end of forty-eight hours. Child seemed lazy and would not try to make its bowels move, even though mother tried in various ways to persuade him. Passed considerable gas and always had a prominent abdomen. Did not seem quite as active as other children. Appetite very good and slept well.

Physical Examination.—Well developed and well nourished male child, 4 years of age. Eyes showed slight conjunctival redness, otherwise negative. Nose normal. Teeth, gums and throat normal. Glands not palpable. Thymus and thyroid negative. Heart showed no enlargement or irregularity or murmurs, and sounds were of good force and intensity. The second pulmonic

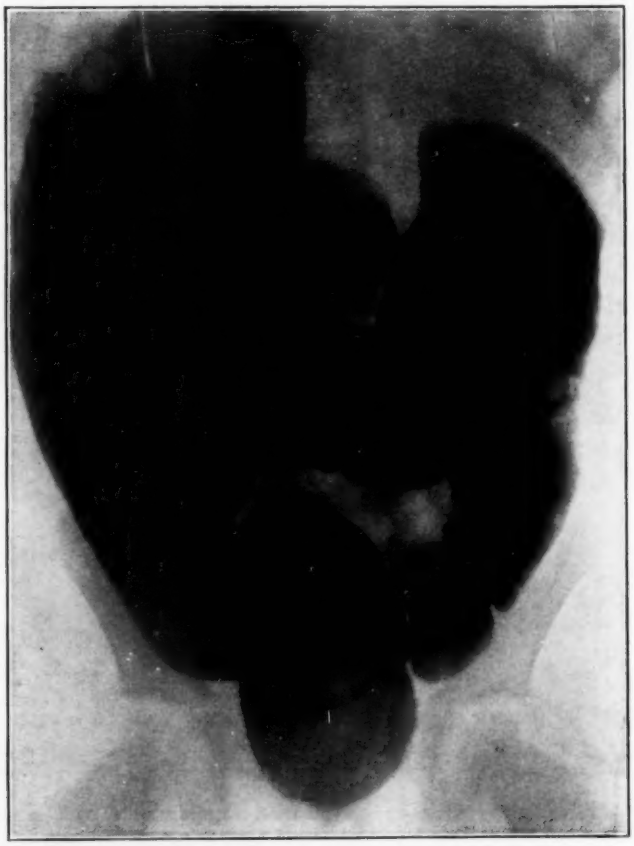

Fig. 1.—Showing marked improvement in case of megacolon after treatment. Note difference in size of lumen of colon.

sound was accentuated. The lungs were negative to auscultation and percussion. The abdomen was moderately distended. No masses, free fluid or tenderness present. Liver and spleen not enlarged. Skin rather pale. Muscles flabby and of poor tone. Reflexes normal. Genitals negative. Rectum negative. Urine was negative for albumin, sugar, acetone and diacetic acid. There was a slight trace of indican present. The sediment was negative. The stool was mushy in character, foul and remnants of food were present. Microscopically, many granules were present. The von Pirquet reaction was negative. The

Wassermann reaction was negative. The child was given a bismuth enema and a roentgenogram of the colon was made. The diagnosis submitted by the roentgen-ray department was: megacolon—large redundant sigmoid.

The patient was put on the following starch-free diet: (The juice of one orange one-half hour before breakfast):

Breakfast: Well done, lean, crisp bacon; one soft boiled egg; 8 to 10 ounces of milk.

Dinner: Chop, fish, chicken or scraped beef; 2 to 4 tablespoons of junket; 2 to 4 tablespoons of custard; 8 to 10 ounces of milk.

3 p. m.: Glass of milk or 2 or 3 ounces of ice cream.

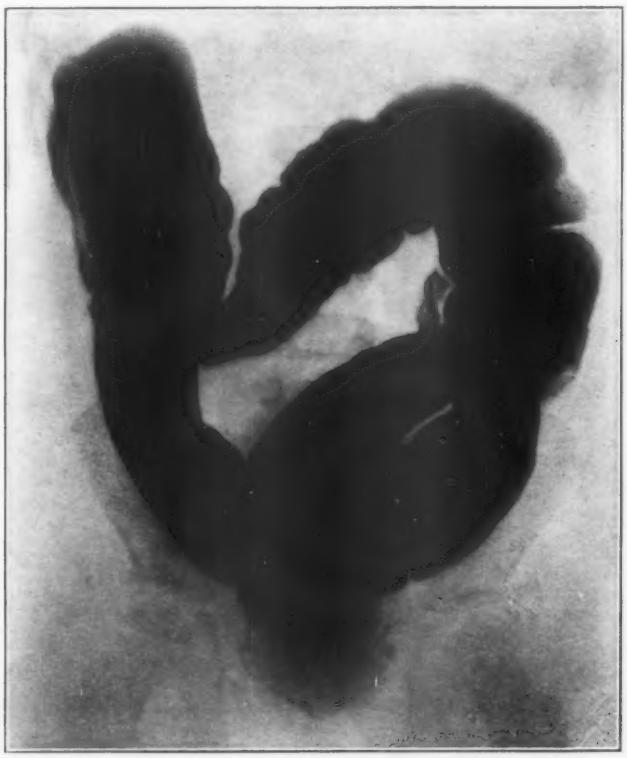

Fig. 2.—Showing a redundant and looped sigmoid in a case of persistent constipation.

Supper: 2 to 4 tablespoons of cottage cheese; 2 to 4 tablespoons of gelatin; 8 to 10 ounces of milk.

(Total number of calories in the above diet, about 1,400.)

In addition to the above diet, the child was given 5 drops of atropin sulphate solution, each drop containing $\frac{1}{1,000}$ grain, three times daily. This was increased one drop daily.

Subsequent History.—In two weeks, mother reported a decided improvement in the constipation, by which time the child was receiving 10 drops of the atropin solution, three times daily. Vegetables and fruit were gradually added to the diet and the atropin solution was reduced. The following note was

recorded March 19: "Condition very good. Only twice in two weeks did mother have to resort to enema. Advised to decrease atropin."

April 3, the mother reported that the child had a normal movement every other day, though large; yesterday had three movements normally. Now getting 6 drops, three times a day. Weight, 17.6 kilos (slight increase). April 10 a second roentgenogram was taken, this showing a decided decrease in the size of the lumen of the colon. Constipation again set in because of the impaction of the bismuth in the rectum. The atropin was again instituted until the bowels began to function normally and daily. July 8, the mother reported that the child had had a normal movement daily for two months, and that she believed that her child was cured. A third roentgenogram was taken, and the decrease in size of the lumen was so noticeable that the roentgen-ray department returned the plate with simply "redundant sigmoid" as the diagnosis.

Comment.—Here is a case of dilatation of the colon (megacolon) diagnosed both clinically and with the aid of the roentgen ray. Constipation dates from infancy and has been persistent. All sorts of drugs were tried, but no attention was given to the preponderance of starch in the diet. Assuming that spasm was playing a rôle in this condition the child was put on increasing dosages of atropin, starting from five drops of a 1/1,000 solution until the child complained of dryness of the mouth. Gradually, well done toast, cooked fruit and green vegetables were added to the diet and the atropin was decreased. The constipation ceased after the spasm was relieved and normal defecation followed.

CASE 2 (52933).—Father living and well. Mother living and suffers from bronchial asthma. No other children. None dead. No miscarriages. No tuberculosis, cancer, rheumatism or nervous diseases in the family.

Past History.—Child was born at full term and delivery was normal. Birth weight, 8 pounds. Breast fed for eight months. Sat up at 8 months. first tooth at 6 months; walked at 16 months; talked at 18 months. Children's diseases: Measles at 3 years of age. Later had "kidney trouble" and was sick for two weeks. Eyes were swollen. No symptoms since except occasional scalding of the urine. No enuresis, but child gets up to pass urine during the night. Appetite fair. Sleep is restless.

Present Illness.—Constipation since six weeks of age, since which time child has never had a normal movement of the bowels. Various cathartics and enemas were constantly given. Child was never allowed to go longer than thirty-six hours without a movement. Passes considerable gas and abdomen always prominent.

Physical Examination.—Well developed and nourished girl 4 years of age. With the exception of slightly hypertrophied and cryptic tonsils and a very prominent abdomen, the physical examination was negative. Weight, 35 pounds; height, 41 inches. Urine negative for albumin, sugar, acetone and diacetic acid. Examination of stool showed a moderate amount of undigested starch; was foul, mushy and showed some evidence of fermentation. A roentgenogram of the colon showed a redundant and looped sigmoid (Fig. 2).

Treatment.—The child was put on a starch-free diet and given 5 drops of the atropin solution as above, to be increased one drop daily. Fruit juices were allowed both morning and night to offset the constipation usually present with a starch-free diet.

Subsequent History.—The constipation did not improve until the child was receiving 9 drops of atropin solution three times daily. Cooked fruits and vege-

tables were added to the diet, and the atropin solution decreased. After three months the mother reported that the child was having a normal bowel movement every day, having discontinued the atropin several weeks before, and was now on a mixed diet.

CASE 3 (51246).—Father and mother living and well. One brother living and well. One miscarriage (spontaneous). No tuberculosis, rheumatism or nervous diseases.

Past History.—Child was born at full term and delivery was normal. Was breast fed for one year, then given a general diet. First tooth erupted at six months. Walked and talked fairly early. Children's diseases: scarlet fever, "summer complaint," pneumonia. Appetite always good. Bowels always constipated. Gets colds easily.

Fig. 3.—Showing slight redundancy of sigmoid, and moderate dilatation of rectum in a case of persistent constipation.

Present Illness.—During the last two years has had frequent attacks of high fever accompanied with a cough. Bowels have been constipated since child was 2 months of age, and would only move when cathartics and enemas were given. Mother has never allowed the child to go longer than forty-eight hours without a bowel movement. Stools are usually hard.

Physical Examination.—Well developed and well nourished boy 6 years of age. With the exception of a rather pendulous abdomen, somewhat distended, and an acute phimosis, the physical examination was negative. The von Pirquet reaction was slightly positive. The urine was negative for albumin, sugar, acetone and diacetic acid. Examination of the stool showed it to be mushy in character, foul and not well formed. Microscopically, there was a moderate amount of undigested starch. The child was given a bismuth enema and a roentgenogram was taken of the colon. This showed a moderate amount of redundancy of the sigmoid and a very large rectum.

Treatment and Subsequent History.—The child was put on a rigid starch-free diet for a few days. Fruit juices were allowed. Atropin sulphate solution was given, starting with 5 drops, three times daily. Improvement was noted one week after treatment was instituted. Additions were made to the diet, and after two weeks the atropin solution was discontinued. The last notation on the history was as follows: "Doing exceedingly well and bowels move normally every day. Child is given raw and cooked fruit, such as peaches, pears, applies, apricots and plums. Appetite is very good. Diet contains only a slight amount of starch."

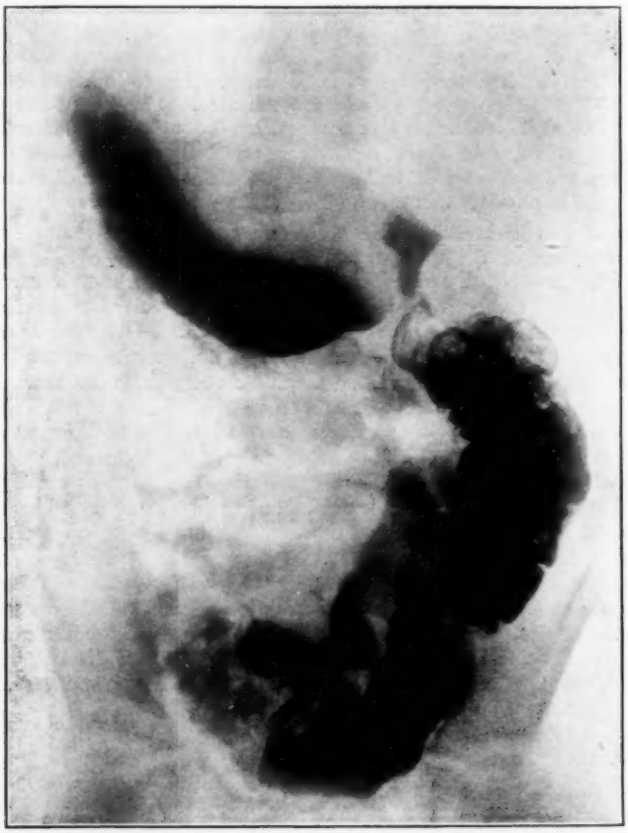

Fig. 4.—Showing redundancy of sigmoid extending up to transverse colon. Figures 1 and 6 are of the same case.

CASE 4 (30465).—Father living and well. Mother living, suffers from headaches. No tuberculosis or exposure. No history of rheumatism, cancer or nervous diseases. No miscarriages.

Past History.—Child was born at full term after a rather difficult labor. Because of sickness following immediately after delivery, mother was unable to nurse it. Was fed on cow's milk for one month, then changed to Eagle Brand condensed milk which it was fed for one year. Early digestion was fairly good. Sat up at 6 months of age, walked at 13 months and talked at 15 months. Children's diseases: Croup at 1 year of age, which has persisted

off and on up to the present time. At 3 years of age child began to have vomiting spells, lasting for four or five days. It was projectile in character and usually associated with constipation. Is never without a cold or cough of some sort. In 1917, patient contracted scarlet fever; temperature 104 F. Urine always negative. Following this infection child has always been constipated and has been compelled to use cathartics and enemas. Has considerable gas.

Present Illness.—About once a month patient has a vomiting spell which lasts from one to four days. Bowels are always constipated and cathartics are used to keep the bowels free. In the interim the child seems to be in fairly good health and comfortable. There is some pain connected with the vomiting.

Physical Examination.—Well developed and nourished child, 9 years of age. Seems to be of a nervous temperament. Parietal bosses very prominent. Eyes and nose negative. The teeth are in good shape. The tonsils and adenoids are hypertrophied. The posterior glands are palpable. The heart and lungs are negative. The abdomen is very prominent, but no masses, fluid or tenderness present. Liver and spleen are negative. The child had an acute phimosis, and circumcision was advised to be done when the tonsils and adenoids were removed. (This physical examination was made two years ago.)

Treatment and Subsequent History.—The vomiting spells and constipation have persisted up to the time of child's visit to the clinic in August, 1919. The physical examination at this time was as follows: Child somewhat undernourished; middle right turbinate enlarged; teeth irregular; tongue coated and papillae very prominent; tabs of tonsillar tissue in fossae; submaxillary and cervical glands are palpable; apex of heart 8 cm. to the left of mid-sternal line, in the fourth space. there is a soft systolic murmur at the apex, not transmitted; the second pulmonic is greater than the second aortic; abdomen is negative; skin is dermographic; reflexes normal. Pulse 96; pressure 85 (systolic), and 65 (diastolic). The blood showed nothing abnormal. Examination of the stool showed considerable free starch. The urine was negative for albumin, sugar, acetone and diacetic acid. The child was given a bismuth enema and the colon was screened. (Through a misunderstanding no roentgenogram was taken.) The roentgen-ray report was as follows: "Moderate redundancy of sigmoid; moderate dilatation of rectum." A complete gastro-intestinal series was made a few days later. After six hours, one half the bismuth remained in the stomach, with the head of the barium column in the colon. The spleen was enlarged. The splenic flexure was distended with gas. The pyloric sphincter was thickened. Conclusion: Constipation and splenomegaly; marked gastric stasis unexplained except by thickened pylorus. The child was put on a starch-free diet and given the atropin solution as above. In two weeks the mother reported the condition much improved Toast and baked apple were added to the diet. Two weeks later the child continued to improve. The last note on the history was as follows: "Child's improvement quite noticeable. Has had no vomiting attacks. Bowels move every day, the amount being small the first day but quite large the following day. Appetite and sleep very good. Vegetables allowed. Has had no vomiting attacks for two and one-half months. Advised mother to continue with the atropin solution because of the pylorospasm."

CASE 5 (54305).—Father and mother living and well. One brother living—has stomach trouble. No children dead. No miscarriages. No tuberculosis, rheumatism or nervous diseases in the family.

Past History.—Child was born at full term and delivery was normal. Breast fed. At 1 month of age had a slight intestinal upset, since which time child has always been constipated. Sat up at 8 months; first tooth erupted at 8 months; walked at 13 months. Has never had any of the infectious diseases. Tonsils and adenoids were removed one year ago.

Present Illness. — Persistent constipation since infancy. Mother has tried various measures for relief, but the child is unable to move its bowels without the aid of a cathartic or enema. Has always been more or less under par.

Physical Examination.—Well developed and well nourished child, 5 years of age. With the exception of a very promient abdomen, the physical examination was negative. The urine was negative for albumin, sugar, acetone and diacetic acid. Examination of the stool showed considerable undigested starch. Roentgenogram of the sigmoid showed redundancy of the sigmoid and dilatation of the rectum.

Fig. 5.—Showing redundancy of sigmoid and dilatation of rectum in a case of persistent constipation.

Subsequent History.—This patient was put on a starch-free diet and the atropin solution. The result of the treatment in this case is not known, as the patient did not return to the clinic, and it seemed impossible to get in touch with them.

CASE 6 (33048).—Father living (is sick—cause unknown). Mother living and well. No other children living. None dead. No miscarriages.

Past History.—Child was born at full term and delivery was normal. Birth weight, 7½ pounds. Breast fed for three months. Early digestion seemed good, but child was always constipated. Had measles at 2 years of age. Later had varicella. Has frequent colds.

Present Illness.—Constipation since child was a baby. Mother has tried massage over the sigmoid, as well as cathartics and enemas, but condition has not improved. Appetite is variable. Mother thinks child's abdomen is more prominent than normal. Does not seem to have any desire to move its bowels. Diet is an unbalanced one and contains a great deal of starchy food. Stools are usually hard.

Fig. 6.—Showing remains of barium meal six hours after feeding—partial pyloric stenosis and hypertrophy in a child 10 years old. Patient had periodic attacks of vomiting. Roentgen ray of colon showed dilatation of sigmoid.

Physical Examination.—Fairly well developed and nourished female child aged 4½ years. With the exception of hypertrophied tonsils and adenoids, the physical examination was negative. The urine showed a very slight trace of albumin, orthostatic in character; it was otherwise negative. The stool showed an excess of undigested starch, and was foul and mushy. The Wassermann and von Pirquet reactions were negative. The roentgen-ray showed redundant sigmoid extending up to transverse colon.

Subsequent History.—The child was put on a starch free diet and given increasing dosages of the atropin solution, beginning with 5 drops, three times daily. Improvement did not begin until the child was taking 27 drops daily. Vegetables were added to the diet and the atropin was reduced. Two months after treatment was instituted the mother reported as follows: "Condition very good. Bowels move daily and normally. No drops for the last two weeks. Appetite much improved." Urine is negative for albumin. Child has gained one pound in weight.

The child having moved when the roentgenogram was being taken, caused the negative to be blurred just enough to make it unfit for clear reproduction, and therefore has been omitted.

DISCUSSION

From an analysis of the above cases it would seem that all those patients treated were helped materially, and any procedure which helps a patient with a dilated and nonfunctioning colon to have a normal

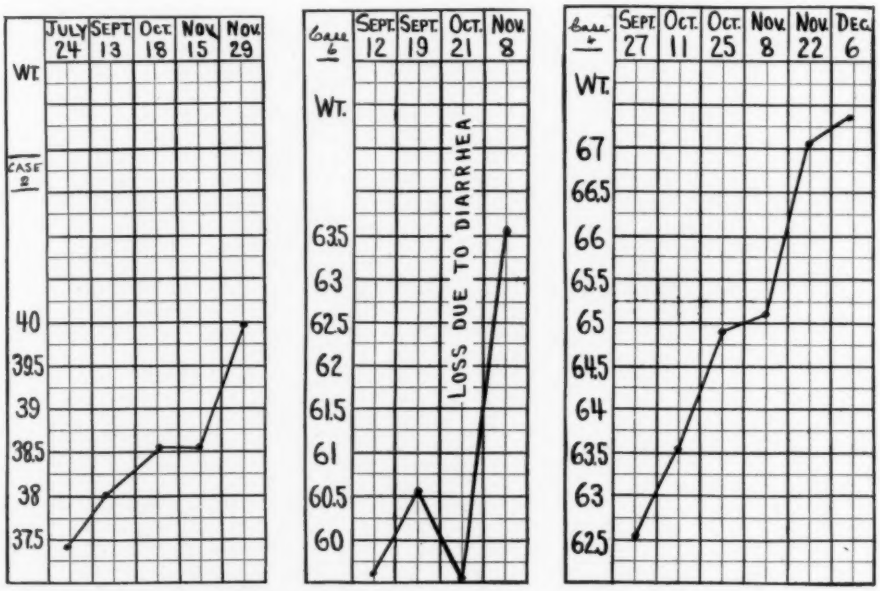

Fig. 7.—Weight curves of three infants following change of diet.

bowel movement every day is worth while. Too often we are confronted by these little ones who have been constipated since birth, and we cheer the mother along by telling her that the child will outgrow the condition, while at the present time she must go ahead with her lubricants, enemas and laxatives. It is also interesting to note that the constipation did not cease until atropin was given. This, I feel, was due to the action of the drug on the smooth muscle fibers of the intestines which were in a state of spasm. Furthermore, after the bowels began to function normally the atropin was stopped and the constipation did not return.

CONCLUSIONS

In conclusion, I would suggest that more roentgenograms of the colon be taken in cases of persistent constipation, especially those dating from infancy. In eight consecutive cases the lower bowel was found to be abnormal and the cause of the condition ascertained. We will at least know more about the etiology even if we cannot help them all medically.

OSTEOGENESIS IMPERFECTA CONGENITA*

H. M. McCLANAHAN, M.D., AND W. W. WILLARD, PH.G.
OMAHA

This condition was first described by Vorlik in 1849. Up to 1906, 140 cases had been reported. So many names have been used in the description of bone abnormalities in infants that an uncertainty remains as to just what is meant in some of these descriptions. For example, Lovett and Nichols,[1] after a careful study of all the case reports, conclude that only fourteen of these are true cases of osteogenesis imperfecta congenita. Since the introduction of the roentgen ray the descriptions have been more accurate. Valuable articles on the subject have also recently been contributed by Schwartz and Bass,[2] Griffith,[3] Hess,[4] Bookman[5] and Mixsell.[6]

Two types are recognized, namely: osteogenesis imperfecta congenita and osteogenesis imperfecta tarda. Authors are convinced that there is a clearly defined distinction between these two types. It is a rare systemic disease of unknown etiology, characterized by imperfect development of bone, numerous fractures and excessive callus formation.

The condition has been described in textbooks under a variety of names as follows: Osteomalacia congenita; rachitis foetalis annularis; malacia myeloplastica; periosteal dysplasia; dystrophia periostale; osteitis parenchymatosa chronica; micromelia annularis; osteoporosis congenita; fragilitas ossium.

Some authors (Griffith,[3] Schwartz and Bass[4]) uphold the theory of an hereditary tendency; others, however, find no familial influences. A study of the recent literature would indicate that syphilis is not a cause. There is no evidence of a disturbance of the endocrine glands, and environment is not a factor.

Opinion differs as to the process of bone formation in this condition. Axhausen says that there is a deficiency in periosteal bone formation due to the lowered function of the osteoblasts, but that the preparatory calcification of cartilage and the absorption of bone are normal. According to Sigowa, the pathologic process consists in a

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* Read before the American Pediatric Society, June, 1919.

1. Lovett and Nichols: Brit. M. J. **2**:915, 1906.
2. Schwartz and Bass: Am. J. Dis. Child. **5**:131 (Feb.) 1913.
3. Griffith: Am. J. M. Sc. **113**: 1897.
4. Hess: Arch. Int. Med. **19**:163 (Feb.) 1917.
5. Bookman: Am. J. Dis. Child. **7**:436 (June) 1914.
6. Mixsell: Arch. Pediat. **34**:756 (Oct.) 1917.

disproportion in the building up and breaking down leading to osteoporosis. Whatever the theory, the fact is that bone formation from periosteum remains in a primary fibrous state. Osteoporosis is a secondary result from the excessive formation of medullary spaces, due to deficient development of bony substance and increased absorption. In the congenital type of case, such as the one described, a more remote cause is embryonal failure of the periosteum and endosteum at the time when ossification should begin, so that the lime salts are deposited in small amounts.

The study of the bone in this case shows evidence that the osteoblasts rising from vascular fibrous bone marrow, because of insufficient nourishment or some toxic influence, did not develop properly, but remained polygonal and later underwent metaplasia into osteoclasts.

REPORT OF CASE

Baby L., male, was admitted to University of Nebraska Hospital, Oct. 1, 1918, aged 3 months. Breast fed up to the time of admission.

History.—Parents in good health. Two older children living and well. No miscarriages. Pregnancy normal and full term. Labor normal. Deformity was apparent at birth. Infant had several fractures, including forearm, thighs and legs. Masses in shafts of long bones easily felt. Head was large and soft. Cry had always been feeble; nursed readily; bowels regular and of yellow color; urine free.

Condition on Admission.—The infant lay on a cot, with thighs flexed on body, seldom moving unless disturbed. The cutaneous reflexes were normal. The abdomen was large, with an umbilical hernia. Spleen and liver were normal. Arms and legs were bent and deformed. Masses of callus could be palpated in the shafts of the humeri, femora and tibiae. Ribs felt soft and pliable, with no discernible fractures. Head felt like a membranous bag with small islands of bone here and there. The skin was soft; the eyes followed objects; no exophthalmos. Heart and lungs were normal; weight 7 pounds and 2 ounces. The mother was certain that the baby had grown, but it was not weighed at birth. Wassermann was negative; von Pirquet tuberculin test was negative. Urine: specific gravity, 1.008; albumin, negative. diacetic acid, negative; acetone, negative; indican, positive; microscopic examination negative.

Blood: Hemoglobin, 70 per cent.; red blood cells, 3,640,000; leukocytes, 18,700. Differential leukocyte count: polymorphonuclears, 56 per cent.; lymphocytes, 40 per cent.; eosinophils, 2 per cent.; special forms, 2 per cent.

As the mother did not remain with the infant, it was placed on a modified milk mixture. The child remained in the hospital from Oct. 1 to Nov. 16, 1918. During the first two weeks of its stay it made a gain of 10 ounces in weight; the next two weeks it lost 8 ounces, due to indigestion and withdrawal of food. For the next three weeks it lost 4 ounces. During the last three days of life, while acutely ill it lost 17 ounces of weight. Ten days before death there developed numerous small furuncles over the body. A culture of the pus revealed *Staphylococcus aureus* in pure culture. While in the hospital, the temperature varied between 99 and 102 F. The bowels were normal, except on two occasions. The urine was free and did not stain. He constantly remained very quiet. The arms were placed in light splints at right angles to the body; the legs were constantly flexed. No attempt was made to apply splints to the legs. November 14, he developed a temperature of 105.6 F. with cough and dyspnea. Respirations were from 60 to 90. The temperature ranged from 103 to 105 F. during entire period of sickness. Examination of

the chest revealed fine râles over the posterior chest with small areas of dulness on percussion. During the last twenty-four hours of life there were frequent attacks of cyanosis, and for the last twelve hours of life there was a constant expiratory grunt. Death from exhaustion, November 18, with a loss in weight of 17 ounces, during this period. As far as can be ascertained from the literature, this is the youngest case with a fatal issue which has been reported in which a careful histologic study has been made.

The conclusions of Professor Willard agree very closely with those recorded by Nichols on an infant dying at the age of 10 months, the youngest previously reported.

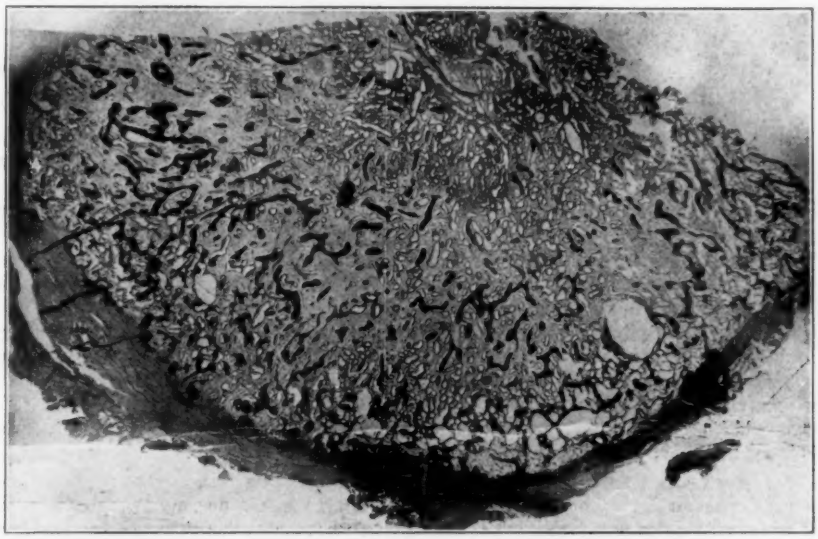

Fig. 1.—Half of transverse section of diaphysis of left femur. Decalcified in nitric acid and formol. Doubly embedded in celloiden and paraffin. Section cut 10 microns thick. Stained in hematoxylin and eosin. Photomicrograph shows an enlargement of 10 diameters. The partially ossified trabeculae are shown black. They are less numerous near the center of the shaft and extend slightly into the periosteum at the periphery. The periosteum is much thicker than normal. It reacts to fracture of the bone by still further thickening, as indicated on the left side of the section. Note the absence of a wall of compact subperiosteal bone which in the normal would extend half way to the center of the diaphysis although the total diameter of the normal is less than half that represented in this case. The blood sinuses of the marrow are the numerous smaller open spaces. Blood cells are adherent to their endothelial walls showing as a darker border to the spaces. The larger spaces in the periphery are possibly shrinkage spaces in the mucoid-like marrow. Blood forming marrow cells are abundant only in the center of the bone where they give a darker appearance to the section.

The apparent shortening and bending of the legs are believed to be due to the pull of the muscles. The relative shortening of the legs and the long body would suggest achondroplasia. However, in the latter condition there is interference with the proliferation of car-

tilaginous cells in the zone between the shaft and epiphysis, resulting in shortening and premature ossification of the long bones. There is also a premature ossification of the sphenoid and the sphenoidal processes of the occipital bone which lead to skull clavis. Both of these conditions are absent, as shown by the roentgenogram and the microscopic studies of the epiphyses. The thickening of the bone is due to increased deposition of cartilaginous cells.

The soft condition of the bone, the multiple fractures and the excessive callus formation combine to make the diagnosis of osteogenesis imperfecta congenita.

REPORT ON NECROPSY

A postmortem examination was made five hours after death by Dr. J. A. Wineberg.

Inspection: Small furuncles were scattered discretely over the head, trunk and extremities. The face was that of an "old man"; the body was emaciated; weight, 5 pounds 15 ounces; the head was large, easily compressed; the arms and legs were short and curved. Callus masses were readily palpated.

Abdomen: No gross changes noted in intestines, liver, spleen or kidneys. No excess of free fluid.

Chest: Both lungs show patches of consolidation with small areas of hemorrhagic infarctions on section. Pleura shows no adhesions. Thymus and thyroid normal. Suprarenals normal. Brain not examined.

Bones: Long bones show multiple fractures with false joints in right tibia and left humerus. Periosteum not ruptured, except on the humerus and on the tibia. On removal of the bones, the left humerus was broken in being dissected out. The bones have a grayish appearance; are easily bent; are soft and pliable. A number of callus rings were present on the site of former fractures.

Microscopic Examination.—The spleen shows congestion. Large areas of necrotic tissue are present in the lungs similar to other postmortem findings in influenzal pneumonia. Kidneys show parenchymatous degeneration. Liver, thyroid, thymus and suprarenals normal. The histologic examination of the bone was made by Dr. Willard.

As the name indicates, this case is one of imperfect development of bone. The interest in the histologic side of the question centers chiefly in the attempt to discover in what way the normal process has been arrested or diverted. This is not easy to do in the absence of stages in the process, the normal of which is an extremely complex and imperfectly understood subject.

However, the outstanding features of normal bone development are well known, involving as they do the early development of an embryonic cartilaginous skeleton which in fetal life is largely replaced by bone and to which are added other bones developed in fibrous tissue and known embryologically as membrane bones.

The histologic structure of all bone tissue is the same regardless of origin, owing to the fact that it is always formed by accretion or deposit of osseous matrix through the combined activity of certain cells derived from embryonic connective tissue. It is thus formed

locally wherever these osteoblasts are active, and when once formed, is fixed, unless absorbed. Such absorption or destruction of bone is a normal part of bone growth and is supposed to be accomplished by large multinucleate cells known as osteoclasts. If this is true, the form and architecture of a bone depends on the balanced interplay of osteoblasts and osteoclasts. The cause behind this is unknown, and must be answered before a real explanation of osteogenesis imperfecta can be advanced.

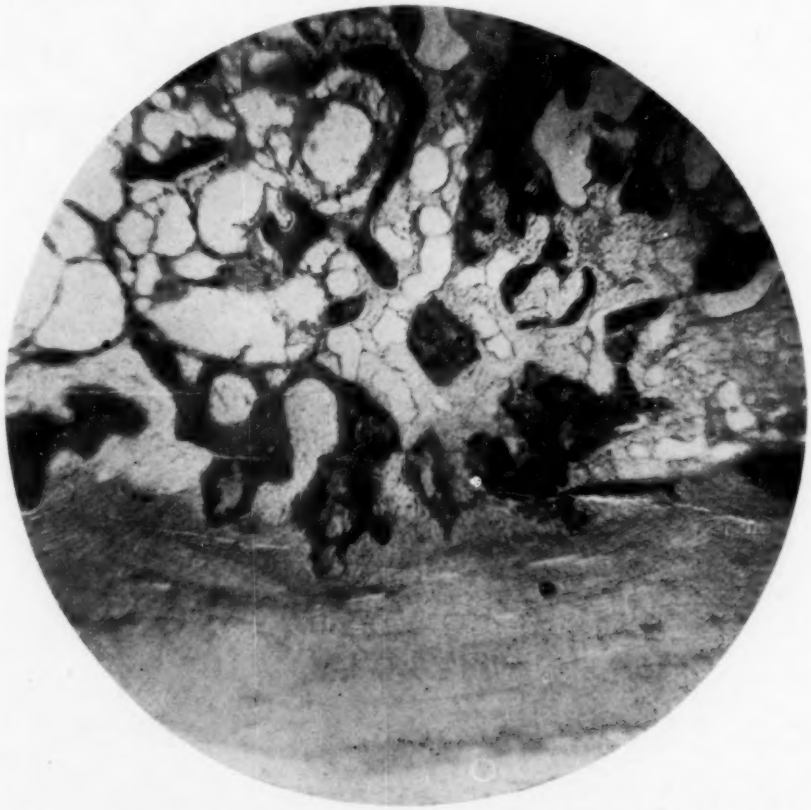

Fig. 2.—Detail from Figure 1. Magnification, 50 diameters. The full thickness of the periosteum with some adjacent bony trabeculae are shown. The darker central areas of the trabeculae are cartilage which has been formed from the periosteum.

Previous investigations on osteogenesis imperfecta indicate that all the bones of the body show much the same changes. We may, then, take the features presented by sections of the femur as typical of this condition. The sections shown in Figures 1, 2 and 3 are from the middle of the shaft or diaphysis of the bone, but they can only be fully understood through a consideration of the bone as a whole. First,

consider the condition of a normal long bone in the new-born child. The shaft is slender but composed of an outer shell of compact bone, which in the case of the humerus is one fourth the total diameter. The marrow cavity is traversed by strong trabeculae entirely of bone. The cartilage of embryonic days has long since been absorbed and no new cartilage is formed. The epiphysis, on the other hand, is composed entirely of hyalin cartilage. The ossification centers that will appear later are preceded at this stage by a vascularization. The region where

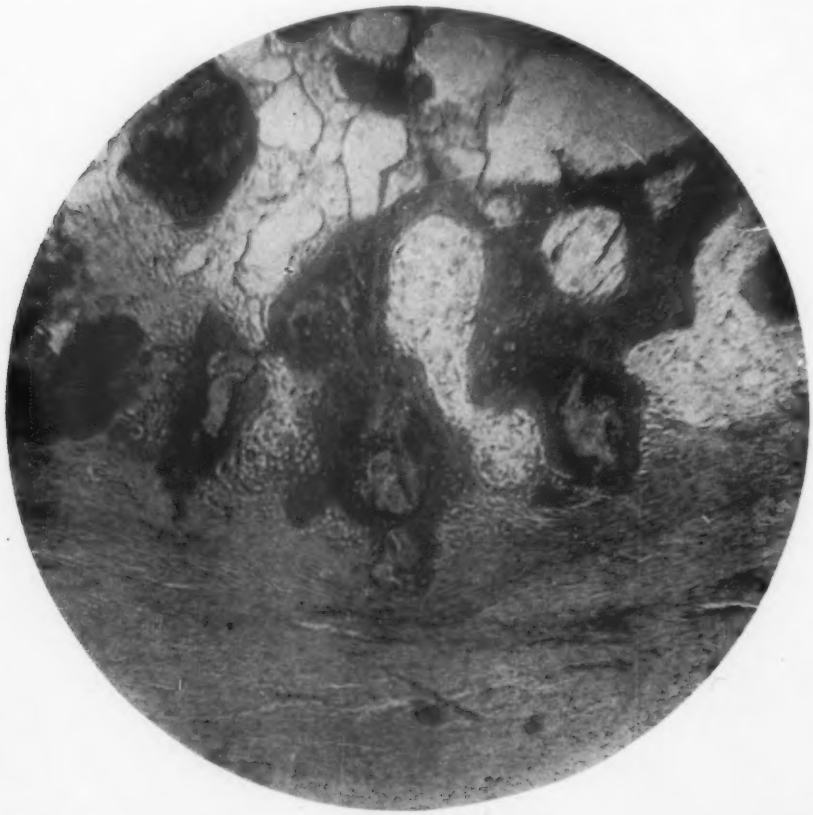

Fig. 3.—Small portion of the field shown in Figure 2. Magnification, 100 diameters. This shows the transformation of periosteal tissue into trabeculae. The encapsulated cartilage cells shown at the left represent the first stage in this process.

the cartilage abuts on the marrow cavity of the shaft is the zone of cartilage absorption and spongy bone formation. As long as the bone is capable of growing in length this zone persists. The increase in length depending directly on a process of cartilage formation in this zone between the epiphysis and the shaft of the bone. Keeping pace

with this cartilage formation on the side of the epiphysis, cartilage absorption is going on next the marrow cavity. This absorption is incomplete in that slender remnants are left which offer the scaffolding on which the osteoblasts of the marrow begin the deposit of bone, thus building up bony trabeculae and contributing the necessary strength to the shaft until the sides of the shaft shall have been strengthened through the formation of a wall of compact bone laid down underneath the periosteum. With the normal growth at the ends of the bone but with the absence of periosteal bone formation at the sides, there would be formed a shaft consisting of a fibrous periosteum enclosing marrow tissue through which anastomosing bony trabeculae would run. If the actual formation of bony tissue around the trabeculae should be still further reduced, they would consist of little more than cartilage remnants with one or two lamellae of bone tissue covering them. If, along with this reduced activity on the part of the osteoblasts or bone forming cells there exists increased or even normal activity on the part of the osteoclasts or bone absorbing cells, there is nothing left in the process to result in bone of any strength whatever.

REPORT OF HISTOLOGIC EXAMINATION OF BONE

A study of the sections in this case of osteogenesis imperfecta shows evidence that the normal processes of bone growth have been diverted in all these ways. All accounts seem to agree that cartilage is formed in the normal way and that the vacuolization of the cartilage leading to the formation of the primary marrow spaces in ossification centers occurs normally but that this process is not met by an immediate and active formation of bony tissue on the part of the osteoblasts. The cartilaginous epiphyses are normal in size and character in this specimen, but the section through the diaphysis as shown in Figure 1 shows complete absence of periosteal bone formation. The periosteum is abnormally thick but there is no bony wall underlying it. The trabeculae scattered through the marrow are slender, and section shows them to be poor in bone tissue. They all show cartilage remnants indicating that bone development has not gone beyond the initial stage. On the other hand, the actual size of the shaft is three times that of normal. Comparison shows that a normal humerus of a new-born child has a slender shaft about 5 mm. in diameter with an enlarged epiphysis of 15 mm. In this case, the shaft of the humerus has a diameter of 14 mm. while the epiphyses corresponds to that of the normal at birth. It should further be stated, that the normal shows a marrow cavity of less than 3 mm. surrounded by a wall of compact bone more than 1 mm. in thickness. The thick stocky form of the bones in osteogenesis imperfecta may be explained as the result of muscle pull on a shaft that has no supporting walls, but histologic study of the periosteum indicates that there is also a process of transformation of subperiosteal tissue into certain constituents of the marrow. This would contribute to the process that widens the marrow cavity. This subperiosteal zone is the one region where abnormal process seems to be an active one. The osteogenic layer of the periosteum, instead of forming orderly circumferential layers of bone, expresses itself in this abnormal condition through the formation of poorly developed type of cartilage. This arises in localized areas and pushing out into the marrow cavity form trabeculae which may become slightly bony as in the case of the other trabeculae. One may recognize in Figures 2 and 3 showing detail of this

region groups of encapsulated cells resembling embryonic cartilage. Between the bases of these trabeculae which extend in from the periosteum multinucleate cell masses often appear. These have the structure of osteoclasts and transitional forms trace their origin to the deeper cell layer of the periosteum. Similar masses are smaller and less numerous throughout the rest of the marrow.

The marrow and its vascularization is strikingly different from the normal. The delicate reticular tissue of normal marrow is replaced by a mucoid-like ground substance containing relatively few cells. Many of these being large fibroblasts, giving tissue a distinctly embryonic appearance. Throughout the marrow are large sinusoidal blood vessels with distinct endothelial lining. The characteristic marrow cells of blood-forming function were found in abundance only in the central part of the section. Careful analysis of these cells could not be made from the material thus far studied, but typical myelocytes, nucleated red cells and megakaryocytes (giant marrow cells) were prominent.

In the foregoing general account there has been no attempt to discuss the literature bearing on the microscopic study of the bones in cases of osteogenesis imperfecta. This will be left to a later study of the cellular elements. This case, as far as presented, agrees in all essentials with the description by Michel and others.

CHONDRODYSPLASIA; MULTIPLE CARTILAGINOUS EXOSTOSES

REPORT OF CASES *

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Until a comparatively recent time, the disease characterized by the formation of multiple cartilaginous exostoses was thought to be a rare affection. Gibney [1] reported the first case in this country in 1875, and in the following year recorded the occurrence of the disease in several members of a family. In 1917, Ehrenfried [2] reported twelve cases and reviewed the literature on the subject. He found that only twenty-nine cases had been reported in America up to that time, and that about forty-two additional cases were mentioned in the discussions. This report stimulated interest in the subject, and case reports during the past two years have been more numerous.

In the cases described, all degrees of associated distortions and deformities of the skeleton are depicted, from the occurrence of simple multiple bony growths causing no particular disturbance, to the occurrence of marked deformities, shortening of the limbs, dwarfing, and associated neurologic symptoms of such severity as to incapacitate the individual. Boggs [3] describes a case of multiple osteochondromata, with great deformity, muscular dystrophy and involvement of the cranial nerves, with blindness.

Ehrenfried found that about 5 per cent. of the reported cases were complicated by malignant osteocartilaginous tumors.

The majority of the cases reported occurred in the period of adolescence and young adult life. A few cases have been reported in children, and Hess in discussing Cowie's [4] paper, refers to an affected family in which the youngest patient was 2 years of age.

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* For permission to study these cases I am indebted to Dr. Roger H. Dennett, Director of the Pediatric Clinic, New York Postgraduate Hospital.

1. Gibney, V. P.: Multiple Exostoses, Med. Rec. **10**:300, 1875.
2. Ehrenfried, Albert: Hereditary Deforming Chondrodysplasia; Multiple Cartilaginous Exostoses, J. A. M. A. **68**:502, (Feb. 17) 1917.
3. Boggs, T. R.: Multiple Congenital Osteochondromata with Degeneration of the Cranial Nerves, and Muscular Dystrophy, Bull. Johns Hopkins Hosp. **24**:310, 1913.
4. Trans. American Pediatric Society, 1917. Abstr. in J. A. M. A. **68**:235, (July 21) 1917.

Three members of a family came to my attention in the outpatient department of the New York Postgraduate Hospital. Two children were brought to the hospital—one a girl, aged 7 years, with a small bony growth on the front of each leg immediately below the knee, and on each arm immediately below the shoulder joint, and a boy, aged 20 months, with a growth on the left scapula. Suspecting the nature of these growths, inquiry was made regarding similar growths in other members of the family, and it was learned that the father was affected, but that the mother and the other daughter, 5 years of age, were not affected.

REPORT OF CASES

CASE 1.—Tina S., white female, aged 7 years, was born in America, of Italian parents.

Family History.—Mother and father are living and well. They are short in stature, but not a disproportionate shortness. The mother has no visible or palpable abnormalities. The father has bony growths on the legs.

Personal History.—Birth was normal. She was breast fed for seventeen months. The first tooth erupted at 6 months; she sat alone at 7 months and walked at 10½ months. She had measles early in life and pertussis at 6 years.

Present Illness.—When the patient was 3 years of age, hard growths appeared on the front of both legs, about four inches below the knees, and on the front of both arms immediately below the shoulder joints. These protuberances grew out rapidly for a short time; but apparently stopped growing a few months later. They caused no trouble and were not painful.

Physical Examination.—On the medial surface of the upper third of the right humerus, a slight enlargment could be seen. Palpation revealed a spur of bony consistency, not tender, and over which the soft parts were freely movable. The spur pointed downward, so that the finger could be hooked under it; it seemed to be about one inch in length and the globular shaped head indicated that it was pedunculated. On the left humerus, in the corresponding area, a smaller bony mass could be palpated. Two growths of the same consistency and about one or one-half inch in diameter were felt on the medial surfaces of both tibiæ, immediately below the tuberosities. Small growths could be felt on both radii, just above the styloid processes, on the clavicles, and several suggestive, though not distinct, enlargements were noted on the phalanges of both hands.

The patient was well developed and well nourished; the head was well shaped and there was no disproportionate shortness of the limbs. The circumference of the head was 19½ inches, and of the chest 21 inches; the height was 43½ inches, and the length of the legs, from the anterior iliac spine to the internal malleolus, was 23½ inches. The middle of the body fell at a point 1½ inches below the umbilicus.

The parents were not aware of any growths, except those on the tibiae and the right humerus. They were causing no trouble whatever and the patient was brought to the hospital only because the protuberance on the arm was noticeable enough to excite comment from those who saw the child.

CASE 2.—Boy, aged 20 months; born in America; a brother of the preceding patient.

Personal History.—Birth was normal; breast fed for nineteen months; he had intestinal disturbances with vomiting and diarrhea early in life, and pertussis when 9 months of age. His first tooth erupted at 6 months; he sat alone at 8 months and walked at 13 months of age.

Present Illness.—A bony enlargement on the back, behind the right shoulder, was noticed when the patient was 5 months of age. It grew out rather rapidly for a few months, then stopped.

Physical Examination.—A mass of bony consistency, about 1½ inches in diameter, could be felt on the right scapula, inferior to the spine. On the third digit of the little finger another growth could be felt. No other protuberances

Fig. 1 (Case 1).—Cartilaginous growths on tibias and femurs.

could be palpated along the accessible parts of the skeleton. The patient was well developed and presented no other abnormalities. The head measured 20 inches, and the chest 19 inches in circumference. The length of the body was 31½ inches, and the middle point was at the umbilicus. The length of the arms was 12½ inches, and the legs 15½ inches.

CASE 3.—John S., aged 29 years; born in Italy; father of the two preceding patients.

Family History.—His mother is dead. Father and one half-brother live in Italy, and two sisters live in America. To the best of his knowledge, no other members of his family have abnormal bony growths.

Physical Examination.—The patient is 5½ feet in height, is well developed and there seems to be no disproportion between body and limbs. A bony mass can be felt on both tibiæ just below the tuberosities.

The only remaining child, a girl, aged 5 years, was submitted to a physical and roentgenologic examination, and was found to be unaffected.

Roentgen-Ray Examination.—Radiographic examination by Dr. William H. Meyer showed multiple osteochondromata on the various long bones, near the epiphyseal lines. The involvement was bilateral in nearly every instance, and the symmetry was marked, although the size of the growths on either side varied considerably in some instances. The bones principally involved were the tibiae, the radii, humerii, femora, clavicles and scapulae. The pelvic bones, the metacarpals and the phalanges were involved to a lesser extent.

Fig. 2 (Case 1).—Spur on left humerus showing the "blown out" lamellar structure.

The part of the bone chiefly involved was in the region of the epiphyseal line, the shaft being comparatively free. There was none of the shortening, curving, and marked thickening of the long bones with globular bubble-like vacuolation at the ends, as was found in the cases described by Ehrenfried. The roentgen ray appearance of the large growths is that of a blown out cystic formation in the lamellar structure, which connects with the lamellæ of the shaft but does not extend up into the shaft for any great distance. The small growths appear as clean-cut exostoses, the defect in the shadow indicating that they are composed of cartilage and bone. The larger growths tend toward spur formation, the spur always pointing away from the nearest joint, a phenomena that seems to be characteristic of this disease.

The most prominent growths on the children were removed and studied microscopically by Dr. Meeker.

PATHOLOGIC REPORT

CASE 1.—Tina S. Nature of specimen: Ecchondrosis.

Gross: Specimen, measuring 18 x 10 x 10 mm., presents a rounded surface showing cancellous bone.

Microscopic: One border of the specimen consists of cartilage with scattered cells in a clear ground substance. Inside this the bone is irregularly deposited with spaces between the trabeculæ. A thickened periosteum extends

Fig. 3 (Case 1).—Pedunculated growth on right humerus.

along the sides of the bone, which is most irregular in outline. The picture is typical of the usual growths on bones of children sometimes associated with rickets.

Diagnosis (Fetal): Ecchondrosis, not yet ecchondroma.

CASE 2.—William S.

Gross: Two pieces, measuring 17 x 15 x 16 mm. and 13 x 10 x 20 mm., respectively. Both are bony in consistency, smooth on one side, which is covered by cartilage, and a cut surface showing cancellous bone.

Microscopic: The picture is the same as the sister's specimen. There is the same irregular atypical change from cartilage to bone, the irregular thickened periosteum with irregular bone outline and deposits of cartilage in the periosteum, all atypical of this form of growth.

Diagnosis: Fetal ecchondrosis, not a true tumor.

The terms ecchondrosis and ecchondroma or chondroma are usually used to define cartilaginous new growths. The former is confined to those simple hypertrophies of preexisting cartilage and the latter to cartilaginous growths in any part of the body. The occurrence of these growths, in the cases described, away from the epiphyses, and on the shaft of the bone and on other bones where cartilage does not exist normally, and the microscopic appearance of deposits of cartilage in the

Fig. 4 (Case 1).—Cartilaginous spur on upper end of right tibia and femur.

periosteum, is evidence that the condition is one of chondrodysplasia, rather than a simple hypertrophy, and the term ecchondrosis is not suitable because of its limitations. Ecchondroma indicates a benign cartilaginous growth anywhere in the body, and fits well with the condition here described.

The term suggested by Ehrenfried, hereditary deforming chondrodysplasia, is not applicable at present to these cases, because there is no deformity, but when we take the ages of these children into consideration, we must recognize the possibility of some deformity later in life, inasmuch as the disease is an anomaly of skeletal growth, and it is probable that the condition is still active and will be until skeletal maturity is attained.

Some of the deformities usually found in these patients are shortness of stature, which is practically constant, even to the extent of

Fig. 5 (Case 1).—Cartilaginous spur on upper end of left tibia and flat growth on femur.

dwarfing, the lack of growth being in the legs, not the trunk, short upper limbs, relative shortening of the ulna and bowing of the radius causing the hand to deviate outward.

The account of Case 4 was obtained from the records of the Children's Mercy Hospital in Kansas City, where the patient was admitted in the service of Dr. C. B. Francisco.

CASE 4.—C. J., white, male, born in Missouri, July 4, 1907.

Family History.—Father died of tuberculosis; two sisters and two brothers dead, cause not stated. The mother is living and well and has had one miscarriage. One sister, living and well, has no bony growths, but the two living brothers have had growths removed from the limbs.

Personal History.—Dec. 2, 1913, he entered the hospital with a deformity of the left arm and a growth under the great toe which caused him to walk on the side of the foot. He was operated on and a large exuberant growth was removed from the ventral surface of the great metatarsal bone. Exostoses were also removed from the anterior and posterior surface of the head of the left radius. Jan. 6, 1919, he reentered the hospital with the statement that the small growths noticed in infancy had steadily increased in size. He had many exostoses, the most prominent were those on the upper ends of both tibiæ, the right external malleolus, and in the left popliteal space. A growth two thirds the size of a lemon was removed from the popliteal space, and one the size of a walnut from the inner side of the femur.

The Wassermann test was positive.

Injury to blood vessels, such as trauma, and aneurysm, due to friction from bony growths, have been reported, especially injuries to the popliteal artery.

The neurologic manifestations noted are ulnar and radial nerve involvement, cord lesions from endostoses, and in one case acromegalic symptoms from a growth in the sella turcica.

NATIONALITY

The nationality of the patients collected by Ehrenfried shows that these cases occurred more often among the Dutch and the Germans, and only once each in an Austrian, a Canadian, an Englishman and an Italian. Two cases occurred in the negro, and Cox's patient is a native of India.

HEREDITY

The heredity feature is well established, and Ehrenfried states that the condition can be transmitted by an unaffected mother, but there is no evidence that the disease can be transmitted by an unaffected male. In the cases reported since Ehrenfried's review, I have been able to find support for this statement. In Cowie's patient, the disease was transmitted by the affected father. In Hess's three patients, the disease was transmitted by the affected father, and in Gorsaline's five cases, a man inherited the condition from the father, and, in turn, transmitted it to two children by his first wife, and five children by his second wife.

Gorsaline [5] mentions a case in a man who has a sister, herself unaffected, but with an affected daughter, thus proving that the unaffected mother can transmit the disease.

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5. Gorsaline, C. S.: Familial Deforming Chondrodysplasia Multiple Cartilaginous Exostoses, Am. J. Roentgenol. **6**:271 (June) 1917.

SEX INCIDENCE

Reincke, in 1891, found that the sex incidence was 3 to 1 in favor of the male, and Ehrenfried, in 1917, found it to be about 5 to 2. Since that time I find that in twenty-nine cases, including my own, the males were affected eighteen times and the females eleven times, a ratio of about 3 to 2.

When we attempt to find an explanation for the high incidence among males, and the transmission of the disease by supposedly unaf-

Fig. 6 (Case 3).—Cartilaginous growths on tibia and fibula.

fected females, it would seem that the affection, if mild, would be more readily discernible on the male body and certainly the male would not be so reluctant to make known the condition and present himself for examination. The wider use of the roentgen ray may bring many more cases to light that would otherwise have escaped attention in the days when most of these cases were reported, and the incidence between the sexes may be more equal. However, this is merely speculative, although there is some evidence of it, in the case reports of the past two years.

AGE

All ages have been mentioned in the case reports from the 2 year old patient of Hess to Frieburg's 80 year old man. The patient in this series, aged 20 months, is, I believe, the youngest in whom the disease has been reported. Also the family here reported represent a very mild degree of involvement.

The disease has very different microscopic changes from those found in osteomalacia, osteogenesis imperfecta, rickets and Paget's disease, although these conditions are mentioned sometimes in their relation to chondrodysplasia.

Its relation to that anomaly of skeletal growth known as chondrodystrophy, however, deserves more than passing notice. In spite of the great physical deformities present in both conditions, the mental development, as a rule, is not retarded.

In his monograph on chondrodystrophia fetalis, Kaufman [6] described three distinct varieties—the hypoplastic, hyperplastic and the malacic. He considered it a dystrophy of the cartilage concerned in the first skeletal formation during the period from the third to the sixth month of fetal life. Some of these patients died in utero, some died in infancy, others in childhood, and some lived to old age, the degree of involvement probably was the factor in determining the fate. In those that lived many variations from the typical achondroplasic dwarf were described. Opie and Alison [7] described the case of a boy, aged 17, who had outgrowths of cartilage, microscopically similar to those found in chondrodystrophy but with no retardation of the growth of long bones. Kaufman also described two fetuses with involvement of the intracartilaginous bones of the head, but with no interference of the growth of long bones. Chondrodystrophy, therefore, may be present without dwarfing, and with widely varying degrees of involvement.

Schorr [8] described a type of chondrodystrophy which makes its appearance at puberty, and to which he gave the name chondrodystrophia adolescentium.

From these descriptions it may be seen that chondrodystrophia exists in a variety of forms, involving those bones involved in chondrodysplasia, and with microscopic changes which are very similar and

6. Osler's Modern Medicine.

7. Opie, E. L., and Allison, N.: Hypertrophic Chondrodystrophy in Infancy and Adolescence; Progressive Anomaly of Osteogenesis, J. Med. Res. **36**:277, (May) 1917.

8. Quoted by Opie and Allison.

seem to differ only in degree, and there is enough similarity in these conditions to warrant the belief that there is a close relationship between them.

1. Both conditions affect bones of intracartilaginous origin without disturbing those of intramembranous origin.

2. In both conditions isolated inclusions of aberrant cartilage within and beneath the periosteum are found. Where more complete microscopic studies can be made on chondrodystrophic subjects, coming to necropsy, these irregular cartilage masses are found widely distributed in fat and in heads of bones.

3. In both conditions there is a progressive proliferation of epiphyseal cartilage, with bone formation at the edge of the proliferations, as is evidenced by the finding of irregular deposits of bone in the cartilage, in the microscopic study of growths removed.

In chondrodystrophy this proliferation of cartilage tends to widen the bone interfering with longitudinal growth, but the proliferation is in an orderly manner giving uniform enlargement, rather than the production of exostoses. The cartilage proliferation is much more excessive in chondrodystrophy, which may account for the gradual and uniform enlargement, as contrasted with the spontaneous isolated outgrowth with the resulting cartilaginous exostoses of chondrodysplasia.

4. The microscopic changes at the epiphyseal line in the two conditions present a striking similarity. In chondrodystrophy the excess of cartilage cells at the sites of ossification occur in irregular groups instead of the normal orderly arrangement in long rows perpendicular to the line of ossification. The line of ossification is straight and very narrow, and instead of a distinct zone of proliferating cartilage cells being present, they are few and unevenly distributed.

Ehrenfried was able to study a specimen from across the epiphyseal line taken from a patient with chondrodysplasia, and he described an excessive growth of intermediary cartilage, with an irregular zone of calcification, and the cartilage proliferating cells scattered in irregular groups instead of forming a distinct zone in orderly arrangement.[9]

SUMMARY

1. Many variations of hereditary deforming chondrodysplasia may occur, from the existence of multiple small cartilaginous exostoses, giv-

9. For an extensive bibliography and a review of the cases reported in American literature, the reader is referred to the article by Ehrenfried.

ing the patient no trouble, to the existence of great deformities, with dwarfing, paralysis, aneurysm and malignancy.

2. The disease manifests itself in infancy.

3. The condition has much in common with chondrodystrophy of infancy and adolescence, and it is probable that a close relationship exists between them.

CALCIUM METABOLISM OF INFANTS AND YOUNG CHILDREN AND THE RELATION OF CALCIUM TO FAT EXCRETION IN THE STOOLS

PART 2.—CHILDREN TAKING A MIXED DIET *

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In a preceding paper on calcium metabolism was considered the absorption of calcium by infants taking modifications of cow's milk and the excretion of calcium in relation to the excretion of fat in their stools. In this paper we discuss the calcium metabolism of older children taking a mixed diet. As stated in the previous paper, by calcium *excretion* is meant the total amount of calcium lost in the stools; whether part of this has been absorbed and is subsequently excreted into the large intestine is not here considered. By calcium *absorption* is meant the difference between intake and the amount lost in the stools.

HEALTHY CHILDREN

The first group considered includes children who were normal as to digestion. Tables 1 and 2 show the calcium absorption in relation to calcium and fat intake for normal children taking a mixed diet; Table 1 including the cases in which the intake of calcium oxid was high, and Table 2 those in which it was low. The values are expressed as grams per kilogram of body weight.

Calcium Absorption and Its Relation to Calcium Intake.—Excluding two unusually high values and one unusually low value, the intake of calcium oxid per kilo ranged from 0.043 to 0.178 gm., with an average of 0.108 gm. In only about one half the cases was the intake more than 0.1 gm. per kilo. The children taking a mixed diet had, on the whole, a much lower intake of calcium oxid per kilo than those who were taking modifications of cow's milk.

The absorption of calcium oxid per kilo also was much lower than was found with the infants; it ranged from 0 to 0.147 gm. of calcium oxid per kilo, the two highest values being found when the intake was exceptionally high.

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* In the article published in the February issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN, p. 112, an error was made in the fourth line of the first paragraph of the Summary. The figure 0.6 gm. should read 0.06 gm.

TABLE 1.—Absorption of Calcium by Healthy Children Taking a Mixed Diet, with an Intake of Calcium Oxid Exceeding 0.09 Gm. per Kilo

No.	Case	Age Yr.	Age Mo.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Percentage of CaO Intake Absorbed	Intake of Fat Gm. per Kg.	Grams CaO Intake per Gm. Fat Intake	Remarks
321	F. S.	1	6	8,975	Constipated	0.244	0.147	60.4	6.8	0.036	Very high calories per kilogram
305	H. F. 1	1	0	7,136	Constipated	0.237	0.115	48.4	4.9	0.048	
307	H. F. 3	1	0	7,749	Constipated	0.178	0.018	10.1	4.7	0.038	
318	D. L.	1	2	8,264	Constipated	0.176	0.067	49.6	4.1	0.043	High calories per kilogram, high proportion carbohydrate
267	O. W. 1	2	6	9,988	Normal	0.175	0.081	46.3	3.8	0.046	
268	O. W. 2	2	7	10,280	Constipated	0.171	0.107	63.1	3.7	0.046	
364	O. W. 4	2	7	10,546	Normal	0.170	0.104	61.4	4.5	0.038	Skimmed milk with corn oil
379	H. F. 4	1	1	8,218	Normal	0.167	0.025	15.2	4.5	0.037	Nut butter
269	O. W. 5	2	8	10,714	Constipated	0.165	0.103	62.5	5.0	0.033	Cod liver oil
308	H. F. 5	1	1	8,455	Constipated	0.163	0.033	20.3	4.2	0.039	Fat-free milk, milk butter
380	H. F. 6	1	2	8,790	Normal	0.157	0.051	32.6	4.0	0.039	Fat-free milk, nut butter
225	C. A. 1	2	3	11,860	Normal	0.156	0.033	14.6	2.9	0.053	Whole milk only. Low total calories
312	R. K. 3	2	0	11,510	Normal	0.153	0.081	52.8	3.3	0.046	
233	F. W. 8	3	7	10,725	Normal	0.149	0.036	24.4	2.9	0.052	Whole milk and cereal. Low total calories
282	J. E.	1	11	10,438	Normal	0.145	0.050	34.4	2.7	0.054	Mostly whole milk. Low total calories
327	R. M.	4	0	12,650	Acid	0.144	0.057	39.4	4.0	0.036	Tuberculous peritonitis. Had cod liver oil
296	E. M. 1	2	3	13,000	Constipated	0.142	0.036	25.5	3.0	0.047	
311	R. K. 2	1	11	11,220	Constipated	0.141	0.036	25.3	2.9	0.052	
313	E. K.	2	0	12,540	Normal	0.140	0.090	64.3	3.0	0.046	
369	W. R. 6	4	9	15,960	Normal	0.137	0.091	66.6	4.0	0.035	Corn oil, considerable milk curd in diet
295	M. M.	5	6	14,777	Normal	0.131	0.016	12.4	2.6	0.050	
309	H. F. 7	1	2	9,170	Constipated	0.128	0.049	38.5	4.3	0.029	
289	M. C.	6	0	14,300	Normal	0.128	0.048	37.1	3.1	0.041	
381	H. F. 8	1	3	9,425	Acid	0.124	0.075	60.7	4.6	0.026	
270	A. W. 1	4	0	14,358	Constipated	0.123	0.061	50.0	2.7	0.046	
317	K. P.	2	8	10,850	Normal	0.119	0.042	34.8	3.2	0.037	Diabetes insipidus. Large proportion carbohydrate in diet
328	D. D.	1	8	11,300	Acid	0.119	0.041	34.3	3.7	0.032	
277	R. L. 3	3	1	13,070	Acid	0.119	0.060	51.0	4.0	0.030	
300	F. B. 1	3	8	15,334	Normal	0.118	0.059	50.2	3.2	0.053	
320	J. O. 2	3	0	15,050	Constipated	0.117	0.064	54.6	2.6	0.046	
291	R. K.	2	1	13,150	Normal	0.114	0.051	44.7	3.2	0.035	
324	T. R.	4	0	15,395	Constipated	0.114	0.047	40.9	2.5	0.046	
366	W. R. 3	4	7	15,700	Normal	0.112	0.037	32.9	4.9	0.023	Corn oil, considerable milk curd in diet
367	W. R. 4	4	7	15,735	Normal	0.112	0.043	38.6	4.9	0.023	Corn oil, considerable milk curd in diet
264	F. W. 9	3	7	11,105	Normal	0.109	0.025	23.1	3.9	0.028	
372	F. W. 6	3	7	11,114	Normal	0.109	0.044	40.5	5.3	0.020	Fat-free milk, corn oil
330	H. F.	4	6	16,130	Normal	0.109	0.024	22.2	2.4	0.046	
373	F. W. 7	2	7	11,365	Normal	0.106	0.036	33.9	5.2	0.020	Fat-free milk, corn oil
276	R. L. 2	3	0	12,879	Normal	0.105	0.012	11.0	3.7	0.028	
287	W. W.	2	5	15,860	Normal	0.105	0.031	29.9	3.2	0.033	
359	R. L. 4	3	1	13,478	Acid	0.103	0.054	52.5	4.7	0.022	Corn oil
326	E. A. 2	7	5	13,025	Constipated	0.096	0.041	42.4	2.6	0.037	Skimmed milk, cod liver oil, rather high proportion carbohydrate
290	D. R.	3	0	14,150	Constipated	0.096	0.025	25.7	3.2	0.030	
322	M. J. 1	3	0	15,400	Constipated	0.093	0.058	62.2	3.1	0.030	
271	A. W. 2	4	1	14,755	Normal	0.092	0.046	50.0	3.3	0.028	

TABLE 2.—Absorption of Calcium by Healthy Children Taking a Mixed Diet, with an Intake of 0.09 Gm. of Calcium Oxid per Kilo or Less

No.	Case	Age Yr.	Age Mo.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Percentage of CaO Intake Absorbed	Intake of Fat Gm. per Kg.	Grams CaO Intake per Gm. Fat Intake	Remarks
297	E. M. 2	2	4	13,100	Normal	0.090	0.026	28.8	2.5	0.036	
301	F. B. 2	3	9	15,795	Acid	0.090	0.010	11.3	2.7	0.033	
272	A. W. 3	4	1	15,334	Normal	0.089	0.050	56.5	3.4	0.026	Corn oil
302	F. B. 3	3	9	15,831	Normal	0.089	0.027	29.8	2.7	0.032	
298	E. M. 3	2	6	13,744	Normal	0.088	0.028	31.4	2.5	0.036	
234	A. L.	3	0	12,440	Normal	0.087	— 0.015	0.0	1.8	0.050	Fever. Low calories per kilogram
299	E. M. 4	2	6	13,979	Normal	0.087	0.018	20.5	2.9	0.030	Skimmed milk, milk butter
383	E. M. 7	2	8	14,040	Normal	0.087	0.004	4.9	2.6	0.032	Fat-free milk, nut butter
274	A. W. 5	4	2	15,725	Normal	0.087	0.012	14.0	3.3	0.026	
382	E. M. 5	2	6	14,175	Normal	0.086	0.020	23.8	2.8	0.031	Skimmed milk, nut butter
254	E. M. 6	2	7	14,130	Normal	0.086	0.001	0.8	2.7	0.032	Fat-free milk, milk butter
248	E. D.	2	6	10,305	Acid	0.084	0.018	22.1	0.9	0.089	No milk except little L. A. milk. High proportion carbohydrate
361	R. L. 8	4	3	14,133	Normal	0.084	0.009	11.0	3.2	0.026	Skimmed milk, nut butter
349	H. J. 1	1	4	10,880	Acid	0.084	0.042	50.6	3.3	0.026	
360	R. L. 6	4	2	14,093	Normal	0.083	0.011	12.8	3.1	0.027	
354	R. L. 7	4	3	14,215	Normal	0.082	0.002	2.6	3.0	0.027	Skimmed milk, milk butter
351	F. B. 5	3	9	14,645	Normal	0.082	0.012	8.5	3.0	0.027	Skimmed milk, milk butter
355	R. L. 9	4	3	14,668	Normal	0.080	0.029	36.4	3.0	0.027	Fat-free milk, milk butter
384	F. B. 6	3	10	14,730	Normal	0.080	—0.007	0.0	2.8	0.029	Skimmed milk, nut butter
362	R. L. 10	4	3	14,815	Normal	0.080	0.005	6.8	3.0	0.027	Fat-free milk, nut butter
385	F. B. 8	3	10	15,243	Normal	0.079	0.021	26.7	2.7	0.029	Fat-free milk, nut butter
365	W. R. 2	4	7	15,412	Acid	0.077	0.025	32.2	4.2	0.018	Corn oil
352	F. B. 7	3	9	15,050	Normal	0.076	—0.001	0.0	2.7	0.028	Fat-free milk, milk butter
353	R. L. 5	4	2	14,115	Normal	0.071	0.031	44.0	2.6	0.025	
350	H. J. 2	1	4	10,880	Normal	0.065	0.012	18.3	2.7	0.024	
325	C. M.	5	0	18,450	Normal	0.064	0.022	33.6	2.0	0.032	
339	E. A. 1	7	0	11,780	Acid	0.061	0.011	18.1	2.7	0.022	Skimmed milk. High proportion carbohydrate in diet
348	M. J. 3	3	4	15,400	Normal	0.055	0.023	42.3	2.1	0.027	High proportion carbohydrate
347	M. J. 2	3	0	15,400	Acid	0.050	0.014	27.3	2.1	0.024	High proportion carbohydrate
337	D. K.	4	0	18,500	Acid	0.048	0.018	38.2	2.5	0.019	
345	E. H.	3	4	14,700	Normal	0.045	0.008	16.7	1.7	0.027	Very low calories per kilogram
336	R. K.	6	0	20,500	Acid	0.044	0.017	39.3	2.3	0.020	Very low calories per kilogram
340	L. S.	3	2	16,000	Acid	0.043	0.013	30.4	1.9	0.023	Very low calories per kilogram
338	R. N.	5	10	20,200	Acid	0.018	—0.001	0.0	1.2	0.015	No milk, high proportion carbohydrate, much vegetable and fruit

The relation of the absorption to the intake is summarized in Table 3. When the intake of calcium oxid exceeded 0.09 gm. per kilo, the absorption in most cases exceeded 0.03 gm. per kilo; the average was 0.055 gm. per kilo. When the intake was 0.09 gm. per kilo or less, the absorption in most cases was less than 0.03 gm.; the average was only 0.015 gm. per kilo. This absorption would hardly supply as much calcium oxid as is normally excreted in the urine by young children. It may, therefore, be inferred that an intake of less than 0.09 gm. of calcium oxid per kilo is insufficient to supply the calcium need of young children taking a mixed diet.

Relation of Calcium Absorption to Fat Intake. — With the older children who were taking a mixed diet, there was great variation in the intake of fat per kilo. On the whole, there was no close relation between the fat intake and the calcium absorption. When the fat intake was high, frequently the calcium intake also was high, and consequently there was good absorption of calcium. The intake generally included less calcium in proportion to the fat than in the case of infants who were taking modifications of cow's milk. In but few instances was there more than 0.05 gm. of calcium oxid per gm. fat in the intake, and in many there was less than 0.03 gm. The highest absorption occurred when the fat intake was greater than 3.0 gm. per kilo and when for every gram of fat in the diet there was from 0.03 to 0.05 gm. of calcium oxid, that is, when the calcium intake exceeded 0.09 gm. of calcium oxid per kilo.

TABLE 3.—Relation of Calcium Absorption to Calcium Intake

Calcium Oxid Intake, Gm. per Kg.	Number of Observations	Absorption 0.09 Gm. or More	Absorption 0.06 to 0.09 Gm.	Absorption 0.03 to 0.06 Gm.	Absorption Less Than 0.03 Gm.
More than 0.09 gm.....	45	7	7	24	7
Less than 0.09 gm.....	34	0	0	3	31
Total.....	79	7	7	27	38

Percentage Absorption of Calcium.—The percentage of the calcium intake absorbed had a very wide range, varying from 0 to 66.6 per cent. When the calcium intake exceeded 0.09 gm. of calcium oxid per kilo, the percentage absorption averaged 40.4; when the intake was 0.09 gm. or less, the average absorption was only 20.3 per cent. of the intake. The excretion of calcium oxid in the stools of healthy children taking a mixed diet did not fall below a certain minimum, whatever the calcium intake. Thus the low intake of calcium oxid did not supply much excess over the amount which was normally excreted in the stools. Hence, the amount available for absorption when the intake was low was much less than the amount which was actually found to

be absorbed when the intake was ample. This explains why, with a small intake, the percentage absorption was lower than with a larger intake, since the excretion tends to remain constant at the expense of the absorption. This observation is paralleled by a similar one in regard to fat, noted in a previous paper,[1] namely, that the percentage absorption of fat was lower when the intake was small than when it was ample.

TABLE 4.—AVERAGE ABSORPTION OF CALCIUM OXID ACCORDING TO WEIGHT

Weight in Kilograms	Number of Observations	Calcium Oxid Intake, Gm. per Kg.	Calcium Oxid Absorbed, Gm. per Kg.	Percentage of Calcium Oxid Intake Absorbed
A. When Intake Exceeded 0.09 Gm. per Kg.				
7- 9	7	0.189	0.068	36.0
9-11	9	0.150	0.072	48.0
11-13	10	0.128	0.046	36.0
13-15	10	0.115	0.044	38.2
15-17	9	0.113	0.051	45.2
Under 11	16	0.167	0.070	41.9
Over 11	29	0.119	0.046	38.7
B. When Intake Was 0.09 Gm. per Kg. or Less				
Under 13	5	0.076	0.014	18.5
13-15	15	0.081	0.013	16.1
15-17	10	0.073	0.020	27.4
Over 17	4	0.044	0.014	31.8

TABLE 5.—AVERAGE ABSORPTION OF CALCIUM OXID ACCORDING TO TYPE OF STOOL

Type of Stool	Number of Observations	Calcium Oxid Intake, Gm. per Kg.	Calcium Oxid Absorbed, Gm. per Kg.	Percentage of Calcium Oxid Intake Absorbed
A. When Intake Exceeded 0.09 Gm. per Kg.				
Constipated	16	0.149	0.064	43.0
Normal	24	0.130	0.048	36.9
Acid	5	0.122	0.057	46.7
B. When Intake Was 0.09 Gm. per Kg. or Less				
Normal	24	0.080	0.015	18.8
Acid	10	0.060	0.017	28.3

Relation of Calcium Absorption to Body Weight.—The absorption of calcium oxid per kilo was to a certain extent related to the total body weight, as is shown in Table 4. With an adequate intake, that is, exceeding 0.09 gm. of calcium oxid per kilo, the children weighing less than 11 kilos absorbed, on the average, about 0.07 gm. per kilo, while those weighing more than 11 kilos absorbed, on the average, only about 0.05 gm. per kilo. The infants, considered in the preceding paper, most of whom weighed less than 7 kilos, had an average absorption of about 0.09 gm. of calcium oxid per kilo. With the children taking a mixed diet whose intake was less than 0.09 gm. per kilo the absorption showed little variation with the weight.

1. Am. J. Dis. Child. **17**:423 (June) 1919.

There was no constant relation between the calcium absorption and the age, irrespective of the weight.

Relation of Calcium Absorption to Type of Stool.—The relation of the calcium absorption to the type of stool is shown in Table 5. The best average absorption was found when the stools were constipated, but the average intake for this group was the highest. Acid stools were rarely found when the intake was over 0.09 gm. per kilo. In

TABLE 6.—EXCRETION OF CALCIUM BY HEALTHY CHILDREN TAKING A MIXED DIET; (1) WITH CALCIUM IN STOOLS EXCEEDING 0.9 GM.

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
295	M. M.	Normal	1.94	38.8	1.70	1.56	0.53	3.1	2.7
225	C. A. 1	Normal	1.85	35.0	1.58	4.10	2.88	17.9	15.3
296	E. M. 1	Constipated	1.84	38.8	1.37	2.29	0.66	4.8	3.6
330	H. F.	Normal	1.76	38.3	1.37	1.62	0.67	4.9	3.8
234	A. L.	Normal	1.08	21.8	1.27	1.81	0.56	4.4	5.2
384	F. B. 6	Normal	1.17	40.9	1.27	2.09	1.07	8.4	9.2
301	F. B. 2	Acid	1.42	43.4	1.26	3.53	1.06	8.4	7.5
307	H. F. 3	Constipated	1.38	36.5	1.24	2.55	1.31	10.6	9.5
313	E. K.	Normal	1.75	37.8	1.24	3.57	1.51	12.2	8.6
233	F. W. 8	Normal	1.60	31.0	1.21	4.10	2.41	19.9	15.1
276	R. L. 2	Normal	1.36	48.1	1.21	1.82	1.00	8.3	7.3
254	E. M. 6	Normal	1.21	37.7	1.20	2.94	1.55	12.9	12.8
311	R. K. 2	Constipated	1.60	31.0	1.18	4.10	1.68	5.8	4.3
352	F. B. 7	Normal	1.16	41.1	1.18	2.85	1.42	12.0	12.2
366	W. R. 3	Normal	1.76	77.0	1.18	4.66	2.26	19.2	12.9
379	H. F. 4	Normal	1.36	37.3	1.17	2.56	1.03	8.8	7.5
274	A. W. 5	Normal	1.36	52.1	1.17	2.56	1.06	9.1	7.8
287	W. W.	Normal	1.67	50.9	1.17	2.40	0.99	8.5	5.9
383	E. M. 7	Normal	1.22	38.1	1.16	2.37	1.04	9.0	8.5
289	M. C.	Normal	1.83	44.5	1.15	1.70	0.71	6.2	3.9
354	R. L. 7	Normal	1.16	43.2	1.13	3.29	1.36	11.9	11.6
327	R. M.	Acid	1.83	50.5	1.11	4.35	1.23	11.1	6.7
308	H. F. 5	Constipated	1.38	35.2	1.10	1.96	1.20	10.9	8.7
362	R. L. 10	Normal	1.18	44.6	1.10	1.68	0.79	7.2	6.7
367	W. R. 4	Normal	1.76	77.0	1.08	3.87	1.35	12.5	7.7
361	R. L. 8	Normal	1.18	45.4	1.05	1.75	0.79	7.5	6.7
324	T. R.	Constipated	1.76	38.3	1.04	1.49	0.55	5.3	3.1
360	R. L. 6	Normal	1.17	43.2	1.02	1.86	0.58	5.7	5.0
351	F. B. 5	Normal	1.20	44.4	1.02	2.74	1.46	14.3	12.1
290	D. R.	Constipated	1.36	44.8	1.01	1.26	0.38	3.8	2.8
232	J. E.	Normal	1.51	28.0	0.99	1.92	1.10	11.1	7.3
302	F. B. 3	Normal	1.41	42.5	0.99	2.25	0.75	7.6	5.3
299	E. M. 4	Normal	1.22	40.1	0.97	2.53	0.90	9.3	7.4
267	O. W. 1	Normal	1.75	37.8	0.94	1.89	1.06	11.5	6.2
380	H. F. 6	Normal	1.38	35.3	0.93	2.24	0.74	8.0	4.9
264	F. W. 9	Normal	1.21	43.6	0.93	2.92	1.45	15.6	12.0
382	E. M. 5	Normal	1.22	39.2	0.93	1.84	0.66	7.1	5.4
300	F. B. 1	Normal	1.81	34.3	0.90	2.16	1.52	16.9	8.4

the few instances when the stools were acid, the intake being adequate, the average absorption was as good as when the stools were constipated or normal. Also, when the intake of calcium oxid was less than 0.09 gm. per kilo, the absorption was practically the same whether the stools were acid or alkaline, but much lower than when the intake was adequate. That the absorption of calcium oxid was dependent more on the amount of the calcium intake than on the type of stool is shown by comparing the values for the normal groups in Table 5. With high intake, the absorption was about three times

as great as with low intake. The influence of the amount of intake of calcium oxid on both the type of stool and the absorption of calcium oxid is further illustrated by three observations on one child, M. J. (Tables 1 and 2). When the intake of calcium oxid was 0.093 gm. per kilo, the stools were constipated and the absorption was 0.058 gm. per kilo; when the intake was 0.056 gm., the stools were normal and the absorption was only 0.023 gm.; again, when the intake was 0.050 gm., the stools were acid and the absorption was only 0.014 gm.

TABLE 7.—Excretion of Calcium by Healthy Children Taking a Mixed Diet; (2) with Calcium in Stools Less Than 0.9 Gm.

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
328	D. D.	Acid	1.34	41.3	0.88	1.63	0.80	9.1	6.0
270	A. W. 1	Constipated	1.76	38.4	0.88	1.43	0.67	7.6	3.8
385	F. B. 8	Normal	1.20	41.8	0.88	1.42	0.41	4.7	3.4
305	H. F. 1	Constipated	1.60	34.9	0.87	1.68	0.90	10.3	5.3
321	F. S.	Constipated	2.19	61.0	0.87	2.46	1.26	14.5	5.8
317	K. P.	Normal	1.29	34.5	0.84	1.23	0.40	4.8	3.1
297	E. M. 2	Normal	1.18	32.6	0.84	1.95	0.69	8.2	5.8
312	R. K. 3	Normal	1.76	38.3	0.83	0.95	0.39	4.7	2.2
291	R. K.	Normal	1.50	42.5	0.83	2.18	1.21	14.6	8.1
298	E. M. 3	Normal	1.21	34.1	0.83	2.53	0.94	11.3	7.8
373	F. W. 7	Normal	1.21	59.4	0.80	3.01	0.44	5.5	3.6
320	J. O. 2	Constipated	1.76	38.3	0.80	1.20	0.68	8.3	3.9
365	W. R. 2	Acid	1.18	64.5	0.80	2.57	0.97	12.1	8.1
325	C. M.	Normal	1.19	36.8	0.79	1.35	0.33	4.2	2.8
277	R. L. 3	Acid	1.55	52.1	0.76	1.18	0.25	3.3	1.6
355	R. L. 9	Normal	1.18	44.6	0.75	3.34	1.08	14.4	9.6
318	D. L.	Constipated	1.45	33.6	0.73	4.04	2.64	36.2	18.2
369	W. R. 6	Normal	2.18	63.1	0.73	1.50	0.44	6.0	2.0
309	H. F. 7	Constipated	1.17	39.8	0.72	2.71	0.73	10.1	6.2
372	F. W. 6	Normal	1.21	59.4	0.72	3.03	0.66	9.2	5.5
326	E. A. 2	Constipated	1.25	34.0	0.72	4.17	2.35	32.9	18.8
364	O. W. 4	Normal	1.79	47.5	0.69	2.10	0.93	13.5	5.2
271	A. W. 2	Normal	1.36	48.1	0.68	1.21	0.50	7.4	3.7
248	E. D.	Acid	0.86	9.7	0.67	4.42	1.83	27.3	21.3
269	O. W. 5	Constipated	1.76	53.5	0.66	3.20	1.69	25.6	9.6
359	R. L. 4	Acid	1.39	63.6	0.66	3.94	1.44	21.8	10.4
268	O. W. 2	Constipated	1.76	38.5	0.65	2.16	1.40	21.5	8.0
339	E. A. 1	Acid	0.72	32.2	0.59	1.51	0.49	8.3	6.8
272	A. W. 3	Normal	1.36	52.1	0.59	2.20	0.89	15.1	6.5
350	H. J. 2	Normal	0.71	29.1	0.58	1.35	0.40	6.9	5.6
353	R. L. 5	Normal	1.00	40.0	0.56	3.57	1.93	34.5	19.3
347	M. J. 2	Acid	0.77	32.4	0.56	4.44	0.41	7.3	5.3
345	E. H.	Normal	0.66	24.5	0.55	1.06	0.55	10.0	5.3
337	D. K.	Acid	0.89	46.6	0.55	1.31	0.07	1.3	0.8
322	M. J. 1	Constipated	1.43	48.2	0.54	1.04	0.31	5.7	2.2
336	R. K.	Acid	0.89	45.6	0.54	1.62	0.21	3.9	2.4
338	R. N.	Acid	0.36	23.9	0.51	1.43	0.40	7.9	11.1
348	M. J. 3	Normal	0.85	31.3	0.49	2.06	0.40	8.2	4.7
340	L. S.	Acid	0.69	30.5	0.48	1.58	0.52	10.8	7.5
381	H. F. 8	Acid	1.17	43.7	0.46	2.07	1.07	23.2	9.1
349	H. J. 1	Acid	0.91	35.5	0.45	1.10	0.35	7.8	3.9

Tables 6 and 7 show for the cases shown in Tables 1 and 2 the calcium excretion in the stools and its relation to the total calcium intake, to the fat intake and to the excretion of total fat and of fat as soap.

Calcium Excretion and Its Relation to Calcium and Fat Intake.—Excluding two exceptionally high values, the total daily excretion of calcium oxid ranged from 0.45 to 1.37 gm., averaging 0.87 gm. Nearly

two-thirds of the values were below 1.0 gm. The excretion was but little related to the intake of calcium oxid. When the intake was low, the excretion did not fall much below the average, so that it formed a much larger proportion of the intake than when the intake was high.

There was no evidence that a very large intake of fat was followed by an excessive excretion of calcium. In fact, no relation was shown between the excretion of calcium and the intake of fat.

Relation of Calcium Excretion to Excretion of Total Fat and of Fat as Soap.—There was but little relation between the calcium excretion and the total fat excretion. Table 6, which gives the instances in which the calcium excretion was greatest, shows only a few more instances of high fat excretion than does Table 5, which gives the instances in which the calcium excretion was lowest.

In order to bring out the relation between soap excretion and calcium excretion, averages were found for the different types of stools, since the average soap excretion varies according to the type of stool. These averages are given in Table 8.

TABLE 8.—AVERAGE EXCRETION OF CALCIUM ACCORDING TO TYPE OF STOOL

Stools	Number of Observations	Intake of CaO, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily
A:					
Constipated	16	1.60	0.90	2.36	1.15
Normal	48	1.38	0.98	2.33	1.00
Acid	15	1.06	0.69	2.45	0.74
B:					
Constipated	16	1.60	0.90	2.36	1.15
Normal—high calcium oxid intake	24	1.63	1.05	2.35	1.10
C:					
Normal—high calcium oxid in stools	24	1.46	1.19	2.59	1.20
Normal—low calcium oxid in stools	24	1.31	0.77	2.07	0.80

Table 8 A shows that in the constipated stools there was a greater soap excretion and a smaller calcium oxid excretion than in the normal stools, although when the stools were constipated, the intake was much greater. Table 8 B, which compares with the group of constipated stools a group of normal stools when the average intake was similar, brings out more clearly the contrast between these two types of stools in calcium and soap excretion.

However, excluding the constipated stools, there was a general correspondence between the variations in calcium excretion and in soap excretion. The average for the acid stools showed a much lower soap excretion, with a much lower calcium excretion, than did the normal stools, but the intake for this group was also much lower than that for the normal group. As was previously noted in the discussion of calcium absorption, the calcium intake apparently influences to a considerable extent the reaction of the stools, a low calcium intake frequently resulting in acid stools.

The relation between calcium and soap excretion is independent of the calcium intake, as is brought out in Table 8 C, in which averages are given for two groups of normal stools arranged according to the calcium excretion. With similar intake the average soap excretion in these two groups of normal stools was exactly proportional to the calcium excretion.

Both with infants fed on milk modifications and with older children taking a mixed diet there was, as has been stated, less calcium excreted in proportion to the amount of soap when the stools were constipated than when the stools were normal. This indicates that there was on the average proportionally less calcium phosphate in the constipated than in the normal stools.

The calcium lost as soap was in most cases an insignificant part of the calcium intake. In only fourteen of the seventy-nine instances in Tables 6 and 7 did it exceed 10 per cent. In the majority of the cases it was less than 10 per cent. of the total calcium *excretion,* and in only eight instances did it exceed 20 per cent.

EFFECT OF VEGETABLE FATS ON CALCIUM METABOLISM

In our study of fat metabolism, one group of children were given corn oil in place of milk fat, and another group nut butter in place of milk fat. The effect of these vegetable fats on calcium metabolism is shown by the averages in Table 9.

TABLE 9.—AVERAGE ABSORPTION AND EXCRETION OF CALCIUM WITH MILK FAT AND WITH VEGETABLE FAT

Kind of Fat in Diet	Number of Observations	CaO Intake, Gm. per Kg.	CaO Absorbed, Gm. per Kg.	Total CaO Intake, Gm. Daily	Total CaO in Stools, Gm. Daily	Fat as Soap in Stools, Gm. Daily
Milk butter	9	0.100	0.016	1.21	1.02	1.36
Nut butter	9	0.100	0.015	1.23	1.06	0.79
Mainly milk fat	35	0.119	0.047	1.52	0.95	0.97
Corn oil	13	0.122	0.052	1.56	0.89	1.40

In the nut butter series the children received in other respects the same diet as during the periods in which the fat given was milk fat. Hence, the findings for these two groups in Table 9 are exactly comparable. There were no control observations to be compared with the observations on corn oil. Accordingly, an average has been found for the cases given in Tables 1, 2, 6 and 7, in which the fat in the diet was mainly milk fat and the intake of calcium was comparable with that of the children taking corn oil.

Table 9 shows that the calcium absorption and the excretion were practically the same whether the fat in the diet was milk fat or vegetable fat. *The only difference in the calcium metabolism was in the*

proportion of the calcium excreted in the form of soap. With the children taking nut butter, the soap excretion was markedly lower than with milk butter and, therefore, a smaller proportion of the calcium excretion could have been bound as soap. With the children taking corn oil the soap excretion was much greater than that of the group of children with similar calcium intake who were taking mainly milk fat. The children taking corn oil received an unusually high intake of fat, so that in respect to fat excretion the two groups are not comparable.

RACHITIC CHILDREN

All the children classed here as rachitic showed, at the time of coming under observation, marked rachitic symptoms, such as inability to stand or walk, distended abdomen and typical bone changes. With most of the children observations were made before treatment was begun, as well as during the period of recovery. Table 10 gives the absorption of calcium oxid per kilo and its relation to the calcium and the fat intake.

Calcium Absorption.—The intake of calcium oxid per kilo was high, in most cases higher than that of the majority of the normal children and in no case was it less than 0.09 gm., which, with normal children, appears to be an adequate intake. In the five observations made before treatment was begun, noted in Table 8 as preliminary observations, the absorption of calcium oxid was low, mostly below the normal average. The highest absorption occurred when the intake was very high. As the condition of the children improved, the calcium absorption increased to well above the normal average, as is shown by the later observations on all the children. With two of the children later observations (371,258) made after recovery was well established, showed a subsequent decrease in absorption. These observations confirm the conclusions of other investigators that *the absorption of calcium is low when active rickets is present, increased above the normal during recovery and falls as the need for calcium ceases to be greater than normal.* It must be remembered that by calcium absorption is here meant the difference between the calcium intake and the amount lost in the stools.

The treatment in our cases consisted in a change of the diet to include more calcium and fat by increasing the milk and giving cod liver oil or milk butter, thus furnishing an extra amount of "fat-soluble A." In the latest observations on F. W. and R. M., made after recovery was well established, vegetable fat replaced butter and cod liver oil.

TABLE 10.—Absorption of Calcium by Rachitic Children Taking Mixed Diet

No.	Case	Age Yr.	Age Mo.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Percentage of CaO Intake Absorbed	Intake of Fat Gm. per Kg.	Grams CaO Intake per Gm. Fat Intake	Remarks
235	L. H. 2	1	6	8,270	Acid	0.288	0.160	55.4	5.5	0.053	Protein milk
265	I. H. 1	2	6	6,560	Normal	0.268	0.228	84.7	8.9	0.030	Cod liver oil, high calories per kilogram
266	I. H. 2	2	8	7,045	Normal	0.248	0.189	76.4	6.7	0.037	Cod liver oil, high calories per kilogram
316	L. R. 2	1	11	8,550	Normal	0.244	0.165	67.5	6.1	0.040	Cod liver oil just discontinued
262*	F. W. 2	3	2	9,207	Normal	0.238	0.059	24.8	3.7	0.065	
315	L. R. 1	1	10	8,205	Normal	0.223	0.127	56.8	4.9	0.046	Cod liver oil just discontinued
257	R. M. 3	2	8	9,916	Normal	0.216	0.113	52.1	6.2	0.035	Butter in place of cod liver oil of previous period. Low carbohydrate
256	R. M. 2	2	7	8,570	Constipated	0.199	0.090	45.2	6.5	0.031	Cod liver oil. Low carbohydrate
261*	F. W. 1	3	1	8,900	Constipated	0.182	0.021	11.8	2.7	0.067	
263	F. W. 3	3	4	10,110	Normal	0.179	0.097	54.1	5.6	0.032	Cod liver oil
304*	R. A. 1	4	0	7,917	Acid	0.176	0.037	26.4	5.3	0.033	
253*	R. M. 1	2	5	8,017	Acid	0.163	0.026	16.1	2.4	0.067	Protein milk and whole milk. Very low calories per kilogram
258	R. M. 4	2	10	10,825	Constipated	0.151	0.068	44.7	4.3	0.035	Olive oil in place of butter of previous period
356	R. A. 2	4	3	9,515	Acid	0.150	0.084	56.3	5.4	0.028	Extra butter
357	R. A. 3	4	4	10,308	Acid	0.138	0.079	57.0	5.6	0.024	Extra butter
333*	L. H. 1	1	5	8,060	Acid	0.126	0.051	40.6	4.2	0.030	
370	F. W. 4	3	6	10,700	Normal	0.113	0.080	71.1	5.6	0.020	Fat-free milk with corn oil
371	F. W. 5	3	6	10,750	Normal	0.113	0.044	38.8	5.5	0.020	Fat-free milk with corn oil

* Preliminary observation.

In the preliminary observations, the percentage of the calcium intake absorbed was with one exception much lower than normal, but during the period of improvement, the percentage absorbed was much higher than with normal children.

The intake of fat per kilo was very high, except in some of the preliminary observations, but, in general, the ratio of the calcium to the fat of the intake was similar to that of the normal children. This resulted from the increase of both fat and calcium in the diet as a therapeutic measure.

TABLE 11.—EXCRETION OF CALCIUM BY RACHITIC CHILDREN TAKING A MIXED DIET

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
262*	F. W. 2	Normal	2.18	33.8	1.64	2.49	1.79	10.9	8.2
235	L. H. 2	Acid	2.38	45.1	1.32	5.71	2.17	20.5	9.1
261*	F. W. 1	Constipated	1.62	24.2	1.43	2.97	1.82	12.8	11.2
304*	R. A. 1	Acid	1.39	42.1	1.10	3.61	2.00	18.2	14.4
253*	R. M. 1	Acid	1.31	19.6	1.10	4.24	1.84	16.7	14.0
257	R. M. 3	Normal	2.15	61.0	1.03	6.42	2.87	27.8	13.3
256	R. M. 2	Constipated	1.70	55.7	0.93	8.59	4.19	45.1	24.7
258	R. M. 4	Constipated	1.62	47.2	0.90	7.34	3.34	37.1	20.5
263	F. W. 3	Normal	1.81	56.7	0.83	3.44	2.05	24.7	11.3
315	L. R. 1	Normal	1.83	39.8	0.79	1.96	1.23	15.6	6.7
371	F. W. 5	Normal	1.21	59.4	0.74	5.30	3.00	40.6	24.8
316	L. R. 2	Normal	2.09	52.5	0.68	3.56	2.29	33.7	11.0
356	R. A. 2	Acid	1.42	51.5	0.62	2.17	0.97	15.7	6.8
357	R. A. 3	Acid	1.42	58.1	0.61	2.33	1.26	20.7	8.9
333*	L. H. 1	Acid	1.01	33.5	0.60	2.62	0.51	8.5	5.0
266	I. H. 2	Normal	1.74	46.9	0.41	1.58	0.64	15.6	3.7
370	F. W. 4	Normal	1.21	59.4	0.35	4.01	1.39	39.7	11.5
265	I. H. 1	Normal	1.76	58.3	0.27	1.49	0.94	34.8	5.3

* Preliminary observation.

Table 11 gives for the rachitic children the calcium excretion in the stools in relation to the calcium and fat intake and to the excretion of total fat and of fat as soap.

Excretion of Calcium.—The range in calcium excretion was about the same as in the case of the normal children. There was no constant relation between the calcium excretion and the intake of either calcium or fat. On the whole, the calcium excretion was not related to the fat excretion. As with normal children, the excretion of soap was sometimes parallel with that of calcium and sometimes not. The lowest values for soap excretion were found when the calcium excretion was lowest, but the highest calcium excretion was not accompanied by the highest soap. In most cases the stools of these rachitic children contained more soap but not more calcium than those of normal children, so that the calcium lost as soap was a distinctly higher proportion of both the calcium excretion and the calcium intake than was found with normal children.

Effect of Treatment.—There was great difference in the calcium metabolism between the period of active rickets and that of recovery. Accordingly, averages are given in Table 12 representing the preliminary and the later observations. Observations 258, 370 and 371 are not included in the average, because they represent periods after recovery was well established and in which the food included much vegetable fat.

TABLE 12.—EXCRETION OF CALCIUM BY CHILDREN WITH ACTIVE RICKETS AND THOSE RECOVERING FROM RICKETS

Condition of Children	No. of Observations	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily	CaO as Soap, Percentage of		Percentage of CaO Intake Absorbed	Percentage of Fat Intake Absorbed
							CaO in Stools	CaO Intake		
With active rickets.	5	1.50	30.6	1.17	3.19	1.59	13.6	10.6	22.0	89.6
Recovering from rickets.....	10	1.83	57.6	0.75	3.73	1.86	24.8	10.2	59.0	93.6

The average calcium excretion was much higher during active rickets than the normal average and during recovery somewhat lower than the normal average. The average intake of calcium oxid was somewhat higher in the period of recovery, making the percentage retention much greater. Although during recovery the fat intake was nearly doubled, the excretion of total fat and of fat as soap was but slightly increased and the percentage absorption of fat as well as of calcium was much improved.

With four of the children, the extra fat was provided by the addition of cod liver oil to the diet. With one child, additional fat was given as butter, with as good result as when cod liver oil was used. Another child showed an improved absorption of calcium on a special "protein milk" in which calcium phosphate and fat were both much increased.

CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION

Table 13 presents the findings for the absorption of calcium per kilo and its relation to the calcium and the fat intake for children suffering from chronic intestinal indigestion.

Calcium Absorption.—The intake of calcium oxid per kilo was in general lower than that of the normal children, and, except in a few instances, the absorption was extremely low. There were only four values for absorption exceeding 0.04 gm. per kilo, and these occurred when the fat intake exceeded 4.0 gm. per kilo. Only once, with as high an intake of fat, was the absorption less than 0.04 gm. per kilo. A high intake of calcium did not regularly result in high absorption,

TABLE 13.—Absorption of Calcium by Children Suffering from Chronic Intestinal Indigestion

No.	Case	Age Yr.	Age Mo.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Percentage of CaO Intake Absorbed	Intake of Fat Gm. per Kg.	Grams CaO Intake per Gm. Fat Intake	Remarks
332	N. C. 1	6	0	8,655	Alkaline	0.242	—0.080	0.0	2.7	0.087	Skimmed milk
331	N. C. 2	6	4	8,705	Alkaline	0.241	0.003	1.4	4.3	0.056	Skimmed milk, cod liver oil
243	H. F. 1	2	10	9,830	Acid	0.202	0.015	7.5	1.7	0.116	Low fat protein milk. Very high proportion carbohydrate
249	S. M.	2	10	7,685	Acid	0.191	0.113	59.2	4.8	0.040	Diet included some L. A. milk
236	G. R. 1	5	0	12,727	Alkaline	0.158	0.032	25.6	1.0	0.158	Mostly fat-free L. A. milk. Very low calories per kilogram
294	M. H.	2	1	7,150	Acid	0.150	0.066	43.8	4.3	0.035	
334	H. L. 1	8	0	14,392	Acid	0.144	0.031	21.7	2.3	0.062	
237	G. R. 2	5	8	14,716	Alkaline	0.140	—0.026	0.0	2.5	0.058	Lactic acid milk, very little solid food
240	F. G. 2	5	5	13,060	Acid	0.133	0.051	37.7	5.7	0.023	Lactic acid milk, cod liver oil
242	F. G. 4	6	5	17,140	Alakline	0.114	0.007	6.2	1.9	0.059	Lactic acid milk, cod liver oil. Very low calories per kilogram
341	A. E.	2	9	5,165	Alkaline	0.110	0.027	24.7	3.0	0.037	Very small child, very little food
244	H. F. 2	3	3	10,000	Acid	0.106	0.047	44.3	4.0	0.027	Protein milk. High proportion carbohydrate in diet
241	F. G. 3	6	0	17,000	Alkaline	0.102	0.014	13.9	3.5	0.029	Lactic acid milk, cod liver oil
255	D. R.	3	0	10,960	Acid	0.089	0.032	36.1	2.2	0.041	Skimmed milk. Very low calories per kilogram
238	G. R. 3	7	8	23,600	Acid	0.083	0.019	22.4	2.1	0.040	L. A. milk, cod liver oil. Very low calories per kilogram
323	E. R. 2	9	4	16,590	Alkaline	0.083	0.014	17.3	2.9	0.028	Cod liver oil. Low calories per kilogram
239	G. R. 4	8	0	22,850	Alkaline	0.081	0.031	37.8	2.4	0.034	L. A. milk, cod liver oil. Low calories per kilogram
278	W. R. 1	4	6	14,650	Acid	0.071	0.004	5.8	2.4	0.030	High proportion carbohydrate in diet
343	H. L. 2	8	0	14,150	Mixed w. urine	0.042	—0.014	0.0	2.1	0.020	High proportion carbohydrate in diet
335	E. R. 1	8	5	12,550	Alkaline	0.041	—0.015	0.0	2.3	0.018	Very low calories per kilogram
342	F. G. 1	5	0	10,508	Acid	0.013	0.004	28.6	2.0	0.007	No milk. Very low calories per kilogram

although in no case was there good absorption when the intake of calcium oxid was less than 0.10 gm. per kilo. The percentage of the calcium intake absorbed was, as a rule, very low.

As has been previously observed, *the absorption of calcium by infants and young children was not good unless the calcium intake bore a suitable ratio to the fat intake.* With normal children, taking a mixed diet, apparently the food should contain from 0.03 to 0.05 gm. of calcium oxid for every gram of fat, and, at the same time, the intake of both calcium and fat should be sufficient. It is seen in Table 13 that in the instances in which the calcium intake was high and the absorption poor, the ratio of calcium to fat in the intake was abnormally high. In other words, there was not enough fat in the food for the amount of calcium. In the other instances of poor absorption of calcium, the intake of both fat and calcium was too low.

TABLE 14.—Excretion of Calcium by Children Suffering from Chronic Intestinal Indigestion

No.	Case	Stools	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily	CaO Possibly Held as Soap, Percentage of	
								CaO in Stools	CaO Intake
332	N. C. 1	Alkaline	2.09	23.5	2.78	3.98	1.58	5.7	7.6
237	G. R. 2	Alkaline	2.06	35.8	2.45	11.20	7.90	32.2	38.3
331	N. C. 2	Alkaline	2.10	37.6	2.07	5.99	1.83	8.7	8.7
243	H. F. 1	Acid	1.99	17.1	1.84	12.50	3.11	16.9	15.6
242	F. G. 4	Alkaline	1.95	33.0	1.83	6.19	3.50	19.1	17.9
334	H. L. 1	Acid	2.07	33.3	1.62	11.34	3.21	19.8	15.5
238	G. R. 3	Acid	1.96	49.0	1.52	7.49	4.05	26.6	20.6
241	F. G. 3	Alkaline	1.73	58.9	1.49	9.54	5.06	33.9	29.2
239	G. R. 4	Alkaline	1.85	55.1	1.15	5.11	1.92	16.7	10.5
323	E. R. 2	Alkaline	1.33	48.4	1.10	14.64	4.72	42.9	35.4
240	F. G. 2	Acid	1.73	74.2	1.06	18.67	5.66	53.3	32.7
278	W. R. 1	Acid	1.04	35.2	0.98	4.34	1.84	18.8	17.7
343	H. L. 2	Mixed w. urine	0.59	29.9	0.79	8.95			
335	E. R. 1	Alkaline	0.51	29.2	0.70	11.20	4.72	67.2	92.4
255	D. R.	Acid	0.97	23.8	0.62	4.68	2.32	37.4	23.9
294	M. H.	Acid	1.07	30.5	0.60	4.79	2.05	34.2	19.2
249	S. M.	Acid	1.47	36.7	0.60	7.30	2.67	44.5	18.2
244	H. F. 2	Acid	1.06	39.8	0.59	3.54	2.07	23.4	18.6
341	A. E.	Alkaline	0.57	15.5	0.43	1.95	1.37	31.9	24.0
342	F. G. 1	Acid	0.14	21.0	0.10	4.68	1.25	125.0	89.3

Table 14 gives for these children the calcium excretion in the stools and its relation to the calcium and the fat intake and to the excretion of total fat and of fat as soap.

Calcium Excretion.—In general, the calcium excretion was much higher than with normal children. When the excretion of calcium oxid was low, the intake was usually very low. There was no constant relation between the intake of fat and the excretion of calcium in the stools. The excretion of fat was in most cases extremely high, only once being less than 3.5 gm. The variation in the excretion of total fat and of fat as soap was entirely unrelated to the calcium excretion.

Since the excretion of soap was large, the calcium lost as soap formed a large percentage of both the calcium excretion and the calcium intake.

As the condition of chronic intestinal indigestion is characterized by the occurrence of very large stools containing a high proportion of fat, there is a temptation to withhold fat from the diet of these children. Our findings indicate that to insure a sufficient absorption of calcium, the intake of calcium must be ample and that it must be accompanied by a suitable proportion of fat. For this reason, it appears to be better to give larger amounts of fat with calcium in due proportion to children suffering from chronic intestinal indigestion, even though the loss of fat in the stools is abnormally great. As was shown previously, in our discussion of fat metabolism, these children retained a much larger amount of fat when their intake of fat was greatly increased, although their excretion of fat was at the same time increased.[2]

EFFECT ON CALCIUM METABOLISM OF VARIATIONS IN CALCIUM AND FAT INTAKE

Table 15 A presents a series of observations on one child, B. W., who first came under observation suffering from osteogenesis imperfecta and malnutrition. The first observation showed with a very high calcium intake an unusually high absorption of calcium. At the time of the second observation, with about the same calcium intake, the absorption had returned to normal. The child was then given 1.6 gm. of calcium acetate daily, equivalent to 0.51 gm. calcium oxid. He received this for twelve days, including the period of observation. This salt had a bad effect on the stools, which became watery, acid and showed evidences of undigested food. The absorption of calcium oxid was unchanged; the additional calcium oxid in the intake was excreted in the stools, there being no increase in the calcium excreted in the urine. The calcium acetate was discontinued, and after the stools had become normal, there was given 2.7 gm. of tricalcium phosphate daily, equivalent to 1.46 gm. calcium oxid. He received this for eight days, including the period of observation. This salt also affected the stools unfavorably, and as in the case of calcium acetate, the excess calcium oxid was all excreted in the stools.

A few months later this child was studied again. He was then considered normal as to digestion. The first observation showed excellent absorption of calcium oxid, rather above the normal average. The fat in the food was then reduced to about one half, and the loss in fat calories was made up by increasing the carbohydrates and the protein. This made the ratio of calcium oxid to fat in the intake

2. Am. J. Dis. Child. **18**:107 (August) 1919.

TABLE 15.—Effect of Variation in Calcium and Fat Intake

No.	Age Yr.	Age Mo.	Weight in Gm.	Stools	Intake of CaO Gm. per Kg.	CaO Absorbed Gm. per Kg.	Percentage of Intake Absorbed	Intake of Fat, Gm. per Kg.	Gm. CaO Intake per Gm. Fat Intake	CaO Intake, Gm. Daily	Fat Intake, Gm. Daily	CaO in Stools, Gm. Daily	Fat in Stools, Gm. Daily	Fat as Soap, Gm. Daily	CaO Possibly Held as Soap, Percentage of: CaO in Stools	CaO Possibly Held as Soap, Percentage of: CaO Intake	CaO in Urine, Gm. Daily	Remarks
A. Observations on B. W.																		
97	1	3	5,873	Softnormal	0.231	.0117	50.8	4.7	0.048	1.36	27.6	0.67	2.54	1.81	27.6	13.3	0.021	Recovering from multiple fractures
279	1	4	6,445	Acid	0.225	0.090	40.0	6.5	0.035	1.45	41.6	0.87	4.33	2.54	29.2	17.5	0.023	Cod liver oil
408	1	5	6,649	Acid	0.319	0.096	29.7	5.7	0.056	2.12	37.7	1.49	8.06	4.76	32.2	22.6	0.017	Calcium acetate (0.51 gm. calcium oxid) given daily. Cod liver oil
404	1	5	6,590	Acid-bad	0.468	0.099	21.2	6.6	0.071	3.08	43.3	2.43	....	....	....	....	.....	Calcium phosphate (1.46 gm. calcium oxid) given daily. Cod liver oil
280	1	9	8,950	Constipated	0.234	0.106	45.4	6.2	0.038	2.09	55.5	1.14	4.57	3.44	30.2	16.5	0.026	Cod liver oil
281	1	10	10,067	Normal	0.246	0.057	23.0	2.7	0.092	2.48	27.1	1.91	3.53	2.25	11.8	9.1	0.034	Low fat, high carbohydrate. Cod liver oil
282	1	11	9,970	Normal	0.180	0.015	7.9	2.3	0.077	1.79	23.3	1.65	2.92	1.97	11.9	11.0	0.046	Low fat, high carbohydrate. Total calories reduced. Cod liver oil
283	2	0	10,888	Constipated	0.174	0.052	30.0	3.9	0.045	1.90	42.1	1.33	3.36	1.86	14.0	9.8	0.020	Proportion of fat increased. No cod liver oil
374	2	1	11,531	Normal	0.174	0.000	0.0	4.3	0.041	2.01	49.7	2.00	6.44	2.06	10.3	10.3	.....	Fat-free milk, corn oil
375	2	1	11,566	Normal	0.174	0.068	39.3	4.3	0.040	2.01	49.8	1.22	7.01	2.42	19.8	12.0	0.047	Fat-free milk, corn oil
377	2	2	11,735	Normal	0.244	0.139	56.8	3.8	0.064	2.87	44.5	1.24	4.65	1.57	12.7	5.5	.....	Chalk mixture (1.0 gm. calcium oxid) daily. Fat-free milk, corn milk
378	2	3	11,777	Normal	0.243	0.166	68.3	3.8	0.064	2.87	44.5	0.91	3.30	1.35	14.8	4.7	.....	Chalk mixture (1.0 gm. calcium oxid) daily. Fat-free milk, corn milk
B. Observations on W. R.																		
367	4	7	15,735	Normal	0.111	0.043	38.6	4.9	0.023	1.76	77.0	1.08	3.87	1.35	12.5	7.7	.....	Corn oil. Milk curd
368	4	8	15,516	Normal	0.276	0.149	54.1	3.9	0.073	4.29	60.1	1.97	4.74	2.58	13.1	6.0	.....	Same diet, less corn oil. Chalk mixture (2.0 gm. calcium oxid) daily

abnormal. The calcium absorption was much reduced, the loss of calcium in the stools becoming much greater, although the excretion of total fat and of fat as soap was less than in the preceding period. The total food was then reduced, involving a considerable reduction in calcium intake. The absorption of calcium became very low and the excretion in the stools, although less than in the preceding period, was still abnormally high. The food was then increased to give an ample and well balanced intake of fat and calcium. The absorption of calcium rose to about the normal average for children taking a mixed diet. The excretion of calcium oxid in the stools was diminished, although still higher than the normal average. The fat in the diet was then supplied entirely in the form of corn oil, and an ample intake of both calcium and fat was provided. The first observation with this diet was made only forty-eight hours after the change. There was a negative balance of calcium and a large excretion of fat in the stools. The next observation, made a few days later, showed normal calcium absorption but no decrease in fat excretion. Later, one ounce of chalk mixture (calcium carbonate), equivalent to 1.00 gm. of calcium oxid, was given daily for six days, including the period of observation. The absorption of calcium was markedly increased, becoming much higher than the normal average; also the fat excretion was diminished. A second observation was made one week later, the chalk mixture meanwhile having been continued. The absorption of both calcium and fat was further improved. Unfortunately, urinary findings were not obtained, so that it is not known whether the extra calcium absorbed was to any extent excreted in the urine.

Table 15 A shows the effect of the administration of chalk mixture with another child, W. R. This child was given 2 ounces of chalk mixture daily, equivalent to 2.0 gm. of calcium oxid for seven days, including the period of observation. As with B. W., the calcium absorption became higher than the normal average, although in this case the excretion also was appreciably increased. More than one half of the added calcium oxid was absorbed, that is, more than was absorbed in the observation on B. W.

To summarize, the points brought out in Table 15 are as follows: High absorption of calcium oxid was found with high intake of calcium oxid; calcium absorption was not increased by administration of calcium acetate or of calcium phosphate; calcium absorption was lowered by reduction of fat intake; calcium absorption was further lowered by further reduction in fat intake and reduction in calcium intake; calcium absorption was raised by increase in fat intake to restore suitable ratio of calcium to fat; calcium absorption was only

temporarily reduced by substitution of corn oil for milk in the diet; calcium absorption was greatly increased by the administration of calcium carbonate in chalk mixture.

PROPORTION OF CALCIUM AND OF SOAP IN STOOLS OF CHILDREN TAKING A MIXED DIET

Table 16 gives the average proportions of calcium and of soap in stools of various types from children taking a mixed diet.

TABLE 16.—AVERAGE PROPORTIONS OF CALCIUM AND OF SOAP IN STOOLS OF CHILDREN TAKING A MIXED DIET

Condition of Children	Stools	Number of Observations	CaO Intake, Gm. Daily	CaO Percentage of Total Solids	CaO Percentage of Total Salts	Fat as Soap, Percentage of Total Solids
Normal	Constipated	23	1.62	8.3	41.8	10.1
Normal	Normal	45	1.38	7.7	39.1	7.9
Normal—high calcium oxid intake	Normal	21	1.66	9.0	42.4	9.2
Normal—low calcium oxid intake	Normal	24	1.13	6.3	36.3	6.5
Normal	Acid	18	1.10	5.1	33.0	5.9*
Normal—high fat diet, much corn oil	Normal	13	1.61	6.0	38.1	9.4
Normal—high fat diet, much corn oil	Acid	4	1.48	4.9	31.7	6.7*
Rachitic	Normal and constipated	10	1.85	7.7	35.9	17.4
Rachitic	Acid	6	1.49	6.1	39.3	10.4*
With chronic intestinal indigestion	Alkaline	10	1.62	8.6	38.6	18.2
With chronic intestinal indigestion	Acid	9	1.48	4.6	34.8	14.0*
Normal—no milk in diet	Acid	1	0.36	1.5	15.4	1.2*
With chronic intestinal indigestion—no milk in diet	Acid	1	0.14	0.6	3.7	7.9*

* Value probably too high since stools were acid.

The calcium percentage of total solids of the stools of children taking a mixed diet was considerably lower than that of the stools similar in water content of infants taking modifications of cow's milk. This is explainable by the presence of food waste, such as cellulose, in the stools of children taking a mixed diet. The constipated stools had a somewhat higher percentage of calcium than the average of all the normal stools. This was related to the calcium intake, as was shown by dividing the normal stools into two groups. The first of these corresponded to an average intake similar to that for the group of constipated stools, and showed an average value for calcium percentage of total solids even higher than that of the constipated stools. The acid stools contained a smaller proportion of calcium oxid than did the normal. Acid stools occurred more frequently when the intake of calcium was low, but difference in intake did not entirely account for the smaller proportion of calcium. This is shown by comparing the average for the group of normal stools corresponding to a low intake with the average for the acid stools. The calcium percentage of total solids was somewhat lower in the acid stools than in the normal

stools corresponding to the same intake. When the intake included large amounts of corn oil the calcium percentage of total solids of the stools was appreciably lower than when milk fat was taken. The total solids of these stools were increased by the large excretion of fat, while the calcium was not increased in proportion.

The normal stools of rachitic children contained a lower proportion of calcium than did those of normal children. With these children also the total solids of the stools were increased by high fat excretion. The acid stools of rachitic children showed a rather high percentage of calcium than the acid stools of normal children, but the intake of calcium was higher with the rachitic children.

The stools of children suffering from chronic intestinal indigestion had a lower percentage of calcium than those of normal children when the intake was similar. The lower percentage of calcium in the stools of these children, also, was due to a much increased proportion of fat.

The stools of two children who had no milk in the food and consequently a very small calcium intake showed a calcium percentage of solids in the stools far below the usual.

With mixed diet the calcium formed a smaller percentage of the total salts than with milk modifications, and the variations corresponded, in general, with the variations in calcium percentage of total solids.

With mixed diet the average soap percentage of total solids was always low and varied with the calcium percentage of total solids. With children taking large amounts of corn oil the soap percentage of total solids was proportionally higher than the calcium percentage. With the abnormal children the soap percentage was markedly higher than with normal children having the same type of stool, while the calcium percentage showed less difference from the normal.

With mixed diet the variations in calcium percentage of total solids of the stools was not related to the water content, since the stools varied little in this respect. Normally, the two factors which mainly affected the calcium percentage of total solids were the calcium intake and the reaction of the stools.

The following summary gives answers based on our observations to the questions on calcium metabolism stated in the preceding paper.

SUMMARY

1. *Normal Absorption and Excretion of Calcium.*—With children taking a mixed diet, the absorption of calcium per kilo was lower than that of infants taking modifications of cow's milk, averaging, when the intake was adequate, 0.055 gm. of calcium oxid per kilo.

The average daily excretion of calcium oxid in the stools of children taking a mixed diet was 0.87 gm.

2. *Calcium Absorption and Excretion in Relation to Calcium and Fat Intake.*—With children taking a mixed diet, the intake of calcium oxid per kilo was lower than that of the infants, the average found for seventy-nine cases being 0.108 gm. per kilo. The absorption of calcium oxid when the intake of calcium oxid was more than 0.09 gm. per kilo in nearly every case exceeded 0.03 gm. per kilo, with an average of 0.055 gm. When the intake was only 0.09 gm. per kilo or less, the absorption rarely exceeded 0.03 gm. per kilo, and in several instances there was negative balance; the average was only 0.015 gm.

The percentage of the calcium intake absorbed when the intake exceeded 0.09 gm. per kilo averaged 40.4; when the intake was 0.09 gm. or less, the absorption averaged only 20.3 per cent.

It may, therefore, be inferred that an intake of at least 0.09 gm. of calcium oxid per kilo is necessary to insure a good absorption by children taking a mixed diet.

The best absorption of calcium oxid occurred when the intake of fat exceeded 3.0 gm. per kilo, and when, at the same time, for every gram of fat there was in the diet from 0.03 to 0.05 gm. of calcium oxid. This is a somewhat lower proportion of calcium oxid to fat than was needed to insure good absorption of calcium oxid by infants taking modifications of cow's milk.

When calcium in the form of chalk mixture (calcium carbonate) was added to the diet, there was a greatly increased absorption of calcium. When calcium was added as calcium acetate or as calcium phosphate the absorption was not increased.

The excretion of calcium was not so closely related to the intake of calcium as in the case of infants taking modifications of cow's milk, and was not at all related to the fat intake.

3. *Effect of Very Small Calcium Intake.*—A very small intake of calcium resulted in either an absorption not much greater than the amount normally excreted in the urine or in a negative balance of calcium.

4. *Relation of Age and Weight to Calcium Absorption.*—The average absorption of calcium oxid per kilo was somewhat lower with the larger children, but the age, irrespective of the weight, had no constant relation to the calcium absorption.

5. *Relation Between Excretion of Calcium and Excretion of Total Fat and of Fat as Soap.*—The excretion of calcium in the stools was not at all related to the excretion of total fat, but bore some relation to the excretion of fat as soap. However, in the constipated stools, which contained the most soap, the calcium excretion was not so great as in the normal stools when the intake of calcium oxid was the same.

6. *Loss of Calcium in Soapy Stools.*—The calcium lost as soap in the stools of normal children taking a mixed diet was, in most cases, an insignificant part of the calcium intake.

7. *Calcium Percentage of Total Solids of the Stools.*—The calcium formed a smaller proportion of the total solids of the stools of children taking a mixed diet than of the stools of infants taking modifications of cow's milk. The calcium percentage of total solids was lower in acid than in normal or constipated stools. The two factors chiefly affecting the percentage of calcium in the stools of children taking a mixed diet were the amount of calcium intake and the reaction of the stools. The *soap* percentage of total solids followed on the average the variation in the *calcium* percentage of total solids.

8. *Calcium Absorption and Excretion in (a) Chronic Intestinal Indigestion; (b) Active Rickets; (c) Recovery from Rickets.*—The *absorption* of calcium by children with chronic intestinal indigestion was extremely low. In the only instances in which the absorption was near the normal both calcium and fat intake were high. The *excretion* of calcium in the stools was very high, except when the intake was unusually low. The excretion of total fat and of fat as soap was very high, but was not paralleled by the calcium excretion.

The calcium *absorption* of children with active rickets was lower than that of normal children, even though the calcium intake was ample. The calcium *excretion* in the stools was somewhat higher than the average excretion in the stools of normal children.

During recovery from rickets, the *absorption* of calcium was higher than the average for normal children. This improvement accompanied the taking of cod liver oil or additional butter with a diet containing an ample amount of calcium. The calcium *excretion* in the stools of children recovering from rickets was lower than that in the stools of normal children.

9. *Effect on Calcium Metabolism of (a) Cod Liver Oil; (b) Vegetable Fats.*—Cod liver oil increased the absorption of calcium, except in cases in which the intake of calcium or of fat was very low.

The substitution of vegetable fats for milk fat did not affect the calcium metabolism of children taking a mixed diet.

A NOTE ON RENAL FUNCTION IN SCARLET FEVER*

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In almost all the studies of renal function in nephritis patients have been utilized in whom the nephritis was an established condition. A few experimental studies have attempted to correlate functional changes with the type of lesion produced by the injection into lower animals of nephrotoxic substances such as uranium or bacillary toxins, and to study in a comparative way the value of various functional tests. While making some functional tests in nephritis in the spring of 1915, it occurred to us that scarlet fever was a condition which offered an exceptional opportunity to study the functional renal changes in nephritis in the human being, from before the onset throughout the various clinical phases of the condition, with particular attention to the time relationship. There is no other disease in which nephritis is such a consistent and looked for complication.

Two types of nephritis must be distinguished in scarlet fever. Early in the course of the disease, in what may be termed the first stage, an acute nephritis, of an interstitial type pathologically, not infrequently develops as a part of the clinical picture of septic infection. This is distinct from the "febrile" albuminuria met with in a very large percentage of cases at the onset of the disease. It is a definite nephritis with casts and albumin in the urine, which, in its presence or absence and in its intensity, bears a direct relationship to the degree of sepsis. As a rule, it is mild in nature and disappears when the streptococcic infection is overcome, but when there is also a necrotic angina, or severe adenitis, or otitis, etc., the nephritis may be an important factor and the kidney shows marked lesions in fatal cases. In the second stage of scarlet fever, near the end of the third week of the disease, the well known "scarlatinal or "postscarlatinal" nephritis develops. The kidney shows extensive glomerular changes and the urine is scanty and contains quantities of albumin, casts and blood cells. Edema is common and uremia occasionally develops. There is no apparent relationship between the two types of nephritis. The first is best explained as a part of the streptococcic infection and the second

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as the result of the toxins of the scarlet fever virus itself. In studying renal function in the nephritis of scarlet fever, the two types must be distinguished carefully, as renal function varies according to the type and degree of pathologic lesion present.

REPORT OF CASES

The two cases used in this study illustrate the two types.

CASE 1 (No. 9918).—A boy, aged 2 years, was admitted to the scarlet fever ward of the St. Louis Children's Hospital, March 11, 1916. From the very onset until death, March 18, the scarlet fever ran a severe septic course with the temperature never less than 102 F., and at times as high as 106 F. The throat was deeply ulcerated, and the tonsils and the mucous membranes of the nose and throat were covered with exudate from which cultures of streptococci were obtained. On the fifth day otitis developed in both ears. The lymph nodes became tremendously enlarged and suppuration took place. Prostration was marked and the patient was usually delirious, but no meningeal complication ensued. Abscesses developed at different points about the tenth day, and a terminal broncho-pneumonia complicated the picture. It was the typical picture of a severe septic infection. Early in the course of the illness the urine showed large quantities of albumin which persisted until the end, and coincidently pus cells and abundant casts appeared.

CASE 2 (No. 9600).—This patient, a boy, 4 years of age, had been at the country department for several months for general malnutrition. About April 11, 1916, he developed a mild scarlet fever with slight angina and adenitis. He was not at all prostrated and had a low fever for a few days. Until April 22 the urine was negative. On that day a trace of albumin was noted, and three days later a few casts were found. April 29 some edema appeared; the urine became scanty and a marked increase in the quantity of albumin was noted. Many casts and red blood cells were found microscopically. All the signs of the acute nephritis increased markedly during the next four days. A gradual improvement followed, slow at first but then with increasing rapidity, until by the end of one month only a trace of albumin and a few casts were left and no clinical sign of nephritis were present. Subsequently the urine cleared entirely.

PLAN OF STUDY

The plan of the study was as follows: Functional tests were to be made on admission. In septic cases with urinary changes these were to be repeated at frequent intervals. In mild cases, with a negative urine early in the course of the disease, weekly functional tests were to be made until either albumen appeared in the daily urine examination, or until any one of the functional tests showed a variation from the normal. The tests were then to be made at frequent intervals. In this way it might be expected to obtain information as to the curve of renal function in uncomplicated cases of scarlet fever, the curve of renal function in developing early and late types of nephritis, comparative data in regard to tests of retention and of excretion, and from these — most important — data as to time relations between functional changes as determined by retention or excretion tests and as shown by

urinary changes. It was simply necessary to study a sufficient number of scarlet fever patients who were suitable for the tests, and sooner or later we would be bound to encounter cases of nephritis. The work was started in the fall of 1915 and continued whenever suitable material was available. It was by no means possible to utilize every patient with scarlet fever, and a large number had to be dropped after one or two of the weekly tests were made. The complete program was carried through in eight cases before one was encountered in which nephritis developed (Case 1), and in nine additional cases before a second case (Case 2) was found. Fortunately, they were frank and marked cases of their respective types. In this way data were collected on seventeen cases of uncomplicated scarlet fever and on two cases of scarlet fever with nephritis. The work was continued as material became available until the spring of 1917, when all laboratory work was interrupted. As it is impossible to continue the work, it has seemed advisable to publish a note on the results so far obtained.

TABLE 1.—Results in Scarlet Fever Uncomplicated by Nephritis. Average for Seventeen Cases

Week	Total Nonprotein Nitrogen, Per Cent.	Phenol-sulphonephthalein Excretion, Per Cent.	Blood Pressure, Systolic
1	25.1	69.3	100
2	22.3	64	97
3	24.2	73	102
4	22.7	71	103
5	23.8	73	103

Tests for renal function, in addition to changes in the composition of the urine, are of two types. In one the ability of the kidney to excrete certain substances, such as dyes, is measured, and in the other the degree of retention or accumulation in the blood of substances normally excreted in the urine is ascertained. The phenolsulphonephthalein test was selected as an example of the first, and the total nonprotein nitrogen was taken as a test of retention. In addition, the blood pressure was determined by the auscultatory method.

RESULTS

In Table 1 the results obtained in the seventeen cases of scarlet fever uncomplicated with nephritis have been averaged. The tests for the first week represent tests made from the second to the fifth day of the disease, the second week from the tenth to the twelfth day, and so

on. In all cases the diet was low in protein and creatinin free. The figures for the nonprotein nitrogen and the phenolsulphonephthalein are all within normal figures and quite uniform throughout the five weeks. Averaging blood pressure figures requires an explanation, as in the younger children the systolic pressure averaged about 90 mm., and in the older 110 mm. The important fact was the level at which the blood pressure remained throughout the course of the disease. This uniformity of figures for the average was maintained in each individual uncomplicated case.

The data for the two nephritic cases are shown in Table 2.

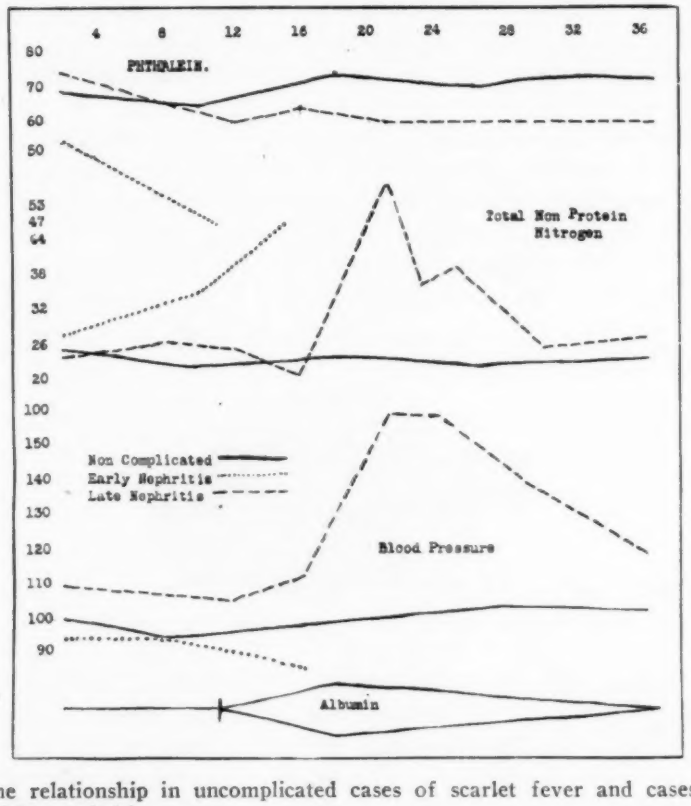
TABLE 2.—RESULTS IN SCARLET FEVER COMPLICATED BY NEPHRITIS

Week	Total Nonprotein Nitrogen, Per Cent.	Phenol-sulphonephthalein Excretion, Per Cent.	Blood Pressure, Systolic
Case 1, Early Interstitial Nephritis			
1	29	52.5	96
2	34	30	94
2½	47	Death ?	92
Case 2, Late Scarlatinal Nephritis			
Date			
4/13	21.5	75	110
4/22	Albumin in urine		
4/23	24.3	60	108
4/25	Casts in urine		
4/27	21	65	115
4/29	Edema		
4/30	39	..	...
5/ 2	53.4	60	115
5/ 4	36.8	..	...
5/ 6	39.3	60	165
5/ 9	29.8	..	...
5/11	25.5	60	138
5/23	28.4	65	110

In order to show the relative time relationship, Chart 1 has been prepared, in which the curve for the uncomplicated cases is shown by a solid line, the curve for fatal case of early interstitial nephritis is shown by the dotted line, and the curve of the glomerular scarlatinal nephritis is shown by a line of dashes.

In the early case there was rapid increase in the accumulation of nitrogenous substances in the blood from the very onset of the disease, and a corresponding decrease in the elimination of phenolsulphonephthalein. Blood pressure was relatively unaffected.

The curve in Case 2, the glomerular type of nephritis, is more interesting. The first sign of urinary involvement was the appearance of albumin in the urine, this change being noted before any changes in the functional tests were manifest. Furthermore, the increase in the albumin and its disappearance from the urine corresponds with the development of the changes in the functional tests. Throughout the course of the disease the elimination of "phthalein" was not affected.



Time relationship in uncomplicated cases of scarlet fever and cases complicated by nephritis.

We have observed, however, in some other cases of post scarlatinal nephritis (established cases which were not followed through their development) a moderate decrease in the phthalein output at the height of the complications. Coincident with the development of the nephritis, a decided increase in the retention of the total nonprotein nitrogen took place. This retention was extremely rapid in development. The curve of increased blood pressure almost parallels the curve of nonprotein nitrogen retention.

What is most important, in our opinion, is that although the functional changes, as noted, occur in the nephritis of scarlet fever and parallel in a fairly close way the urinary changes, the albumin appeared in the urine before the changes in renal function took place. Hence, as a routine measure, the urinary examination for albumin as ordinarily carried out in scarlet fever is of more value than the functional tests in announcing the onset of an impending kidney complication.

AN IMPROVED NEEDLE FOR SINUS THERAPY*

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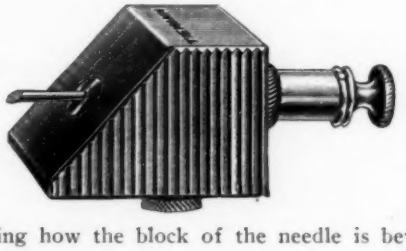
About one year ago [1] I described an apparatus for puncture of the superior longitudinal sinus. This consisted of a needle 4 cm. long, with obturator, which fitted into a rectangular block 3 cm. in thickness, thereby allowing 1 cm. of needle to project. By means of a set screw, the needle was made adjustable to any desired length up to 1 cm. This apparatus has been used extensively in the Babies' Hospital for the intravenous administration of arsphenamin, physiologic sodium chlorid solution, glucose solution and sodium bicarbonate solution, as well as for the withdrawal of blood for diagnostic purposes. A slight modification has suggested itself which greatly simplifies the procedure of sinus puncture and still further minimizes the risk of injuring the wall of the sinus or the cerebral cortex.



Showing how the block of the needle is bevelled.

The original instrument entered the sinus at right angles to its long axis. A miscalculation of the depth of the sinus would make it possible to transfix the wall. For this reason the new apparatus is made to enter the sinus at an angle of 50 degrees with its long axis and pointing to the occiput. This was done by merely bevelling the block at an angle of 50 degrees, as shown in the accompanying figure. It is readily seen that an excessive length of needle in the new apparatus is not likely to damage the wall of the sinus, owing to the oblique direction in which the needle is introduced. The fact that the needle points in the direction of the blood flow is also an advantage.

The needle is adjusted to the desired length and is held in place by the set screw. The block is grasped between the thumb and index

* From the Babies' Hospital, New York.

1. Goldbloom, Alton: Am. J. Dis. Child. **16**:359 (Dec.) 1918.

finger of the left hand and held with the set screw pointing downward. The index finger of the right hand is used to locate the posterior angle of the fontanel. The point of the needle is then placed just over the tip of the right index finger. The finger is then withdrawn and the needle pushed in to the full depth. The obturator is then removed. When this procedure is carefully followed it is most unusual to miss the sinus. When blood does not appear upon withdrawal of the obturator it is usually due to the fact that the needle has not penetrated deeply enough. In such event, with the block still in place, the set screw is loosened and the needle gently pushed in until blood appears. With the modified instrument from 4 to 6 millimeters of needle may be allowed for young infants, and from 5 to 8 millimeters for older ones.

PROGRESS IN PEDIATRICS

REVIEW OF LITERATURE ON RESPIRATORY DISEASES

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INFLUENZA

Etiology.—The concensus of opinion is one expressed by Winchell and Stillman [1] in discussing the relationship of *B. influenzæ* to epidemic influenza. He believes that no matter what the etiologic relationship is, there can be no doubt about its importance as a secondary invader in this type of respiratory infection. The frequency of occurrence of *B. influenzæ* in the throats of normal persons has been as great in the period subsequent to the epidemic as it was during that time. It may persist in the throats of healthy carriers for a considerable length of time. Kolmer,[2] as a result of complement fixation, phagocytosis and agglutination tests, reaches the same conclusion, and shows that *B. influenzæ* played the most important rôle in the disease as an organism of secondary infection, if not the actual or primary etiologic agent. However, no definite proof has been submitted that *B. influenzæ* is the direct etiologic factor, and Lord [3] says that there is no justification for that belief. Nash [4] inquires if the liability of children and young adults is due to a streptococcus infection. If so, the epidemic resembles scarlet fever. If on the other hand, the streptococcus is the infectious agent, is it not strange that the visible changes in the throat are so little marked, for he had not seen a case in which follicular tonsillitis or enlarged cervical glands were present. Park and Williams,[5] as soon as they discovered *B. influenzæ* in large number in the early cases, both in pure cultures from the lungs and in very large numbers, in and between the cells in direct smears from the tracheas examined at necropsy, concluded that whether or not this bacillus is the exciting cause, it gave evidence that it had a marked pathogenic action.

The isolation of the bacillus was no doubt attended by difficulties, and this gave rise to the many conflicting theories as to the direct

1. Winchell, Agnes J., and Stillman, Ernest G.: The Occurrence of *B. Influenzae* in the Normal Throat, J. Exper. M. **30**:497, 1919.
2. Kolmer, J. A., Triest, M. E., and Yagle, E.: Serum Studies on the Etiology of Influenza, J. Infect. Dis. **24**:583, 1919.
3. Lord, F. T., Scott, F. C., and Nye, R. N.: Relation of the Influenza Bacillus to the Recent Epidemic of Influenza, J. A. M. A. **72**:188 (Jan. 18) 1919.
4. Nash, W. G.: The Age Incidence of the Prevailing Epidemic, Brit. M. J. **2**:686 (Dec. 21) 1918.
5. Park, W. J., and Williams, A. W.: Etiology of Influenza, Am. J. Pub. Health **9**:45 (Jan.) 1919.

cause. Stillman [1] had the best results using an oleate hemoglobin agar with a reaction of from p_H 7.2 to 7.5. Bernstein and Loewe,[6] employing gentian violet blood agar, isolated the bacillus comparatively easy, and they claim that with the use of this medium, the percentage of positive results is much higher than when plain blood agar is used.

Clinical Course.—La Fetra [7] admits that the mortality among children was low when compared with young adults, but takes exception to the statement, made early in the epidemic, that infants and young children escaped the disease. More than nine hundred patients were seen in the last quarter of 1918, the great majority having only moderate fever after the first day, a great many having a severe tracheitis. About 10 per cent. had pneumonia, and of these about one half died. Clinically, the cases were divided into several main divisions:

1. Those with high fever and prostration but without physical signs of any localization.
2. Cases with rhinitis or pharyngitis or both.
3. Cases with tracheitis or laryngitis.
4. Cases with bronchitis.
5. Cases with pneumonia.

In the last group, four-fifths were cases of bronchopneumonia. Cyanosis was not as frequently present as in adults. Six patients had interstitial emphysema. In the matter of blood counts there were great variations, but the leukopenia of adults was not characteristic in children.

Achard [8] describes thirty-two cases occurring in children less than 2 years of age. Thirteen of these had pneumonia. He believes that influenza in infants is by no means exceptional, that the forms of the disease may differ and the gravity vary. Infants do not possess any real immunity. Speaking only of general impressions, Locke [9] thinks that the death rate appears to be high for those under 5 years of age, to decrease sharply to the age period of from 15 to 19 years, when it increases rapidly to the maximum, in the period of from 30 to 34 years. The series of pneumonia cases following influenza seen at the Babies Hospital in New York, according to Wollstein,[10] was far more severe,

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6. Bernstein, E. P., and Loewe, L.: A Simple Method for the Isolation of the Influenza Bacillus, J. Infect. Dis. **24**:78 (Jan.) 1919.

7. LaFetra, L. E.: Some Clinical Manifestations of Influenza in Children, Am. J. M. Sc. **157**:770 (June) 1919.

8. Achard, C.: Influenza in Infants, Brit. J. Child. Dis. **16**:78 (April-June) 1919.

9. Locke, E. A., Ronne, G. E., and Lande, H.: The Clinical Aspects of the Recent Influenza Epidemic, Boston M. & S. J. **180**:124 (Jan. 30) 1919.

10. Wollstein, M., and Goldbloom, A.: Influenza in Infants, Am. J. Dis. Child. **17**:165 (March) 1919.

more extensive and more highly fatal than any other group of pneumonia cases observed in the hospital over a similar period of time. Eighteen cases which came to necropsy form the basis of her study. In this series of cases a definite leukocytosis was found in ten, no increase occurred in five and a leukopenia was present in one syphilitic infant. The eighteen cases showed a variety of lesions making a characteristic picture of congestion, edema, general bronchitis, scattered pneumonia lesions differing in age but involving two or more lobes, subpleural hemorrhages and pleural exudate. Capillary hemorrhages in the other viscera were constant.

The influenza bacillus was grown in every case, but not alone. Pneumococcus Type IV was the bacterium most frequently associated with it. During life the sputum contained *B. influenzæ* in thirteen out of seventeen cases in which it was searched for.

Montgomery [11] concludes that the tendency in uncomplicated cases of influenza in infants and children is toward a leukopenia rather than a leukocytosis, and that there is only a slight leukocytosis in complicating pneumonia. In his entire series there was a leukocyte count of less than 10,000 in the fatal pneumonia cases. The differential counts showed nothing that could be of help in either prognosis or diagnosis.

In considering the clinical course of influenza in children, Ostheimer [12] lays particular stress on the uniform drowsiness, even stupor, noticed in all his cases. This, with the fever, was noticed in every case, regardless of age. The next common symptom was loss of appetite, noted in 69 per cent. Some of the older children continued to eat well, in spite of the evidences of toxemia. The aches and pains so commonly described in adults were present in only 27 per cent. — among the older children—as was to be expected. Smith [13] divided his cases into three clinical groups.

1. Temperature elevation with minor symptoms.
2. Temperature elevation with vomiting as the main symptom.
3. Temperature elevation with coryza or cough as the main symptom.

Complications and Sequels. — Many of these, other than pneumonia, have been reported. From a group of children who were suffering from influenza and who had an unusual clinical entity, namely, "influenzal croup," Regan [14] selected twenty for study. From

11. Montgomery, J. C., and Dunham, E. C.: Leukocyte Counts During Influenza in Infants and Children, Am. J. Dis. Child. **18**:153 (Sept.) 1919.
12. Ostheimer, M.: Influenza in Children, Med. Clin. N. Am. **2**:743 (Nov.) 1918.
13. Smith, A. D.: Influenza and Bronchopneumonia in Children, Arch. Pediat. **36**:214 (April) 1919.
14. Regan, J. C., and Regan, K.: Influenzal Croup, Am. J. Dis. Child. **17**: 377 (June) 1919.

the close resemblance of their symptoms to diphtheria many were at first admitted to the contagious ward. Later, because of the constant absence of a membrane, the failure to respond to antitoxin and the uncertain effects of intubation, made it necessary to consider some other etiologic factor than diphtheria. Symptoms previous to admission were the usual ones of influenza. From two to ten days later, the signs of croup appeared. In a few cases the first symptom was a croupy cough, which became progressively worse, associated with symptoms of laryngeal stenosis.

Clinical Course.—This depended on the extent and severity of the laryngeal involvement and the presence or absence of a complicating pneumonia. It was common for the symptoms of laryngeal stenosis to progress to a point of impending asphyxia. When pneumonia is present, the laryngeal stenosis dominates the clinical picture. Certain of the symptoms differ from the croup of diphtheria. There is no membrane, and the mucous membrane is dry and glistening, in contrast to the moist appearance in diphtheria. It is most frequent between three and ten years, is more common in boys than in girls, and the mortality is low, five deaths in a series of twenty cases. Stenosis was present in all cases, and in all but three cases pneumonia occurred.

Klebs-Loeffler bacilli were not found. The bacterial flora was very similar to that noted in influenza cases. From the nasopharynx the Pfeiffer bacillus, the pneumococcus and *Micrococcus catarrhalis* were obtained. In postmortem cultures from the larynx and trachea, the staphylococcus was most constant, the Pfeiffer bacillus and the pneumococcus being next in order of frequency. In cultures from the lung, the Pfeiffer bacillus was predominant, with streptococci and staphylococci present in lesser numbers. A varied blood picture was seen. Leukopenia occurred, but was not constant. A moderate leukocytosis was most constant, as the frequency of pneumonia would lead one to expect.

The nervous system did not escape, but D'Espine [15] saw no suppurative meningeal cases during the epidemic, but describes briefly cases reported by Brat and Fobler in which influenza bacilli were grown from the spinal fluid. The cases with nervous complications seen by him were divided into two groups, one a meningeal form and the other a convulsive or eclamptic form. The former shows distinct changes in the spinal fluid, with apathy and cerebral nerve palsies. The latter show no changes in the spinal fluid, except increased pressure, but are characterized by convulsive seizures and a rapidly fatal outcome. Neal [16] draws attention to three points in reviewing the cases

15. D'Espine: Nervous Complications of Influenza in Children, Arch. de méd. d. Enf. **22**:1 (Jan.) 1919.

16. Neal, J. B.: Meningeal Conditions Noted During the Epidemic of Influenza, J. A. M. A. **72**:714 (March 8) 1919.

of meningeal involvement during the influenza epidemic: (1) Very few cases of meningitis have been due to the influenza bacillus. (2) A larger percentage of cases of epidemic meningitis was seen than is usual. Cultures of the spinal fluid were made in blood as well as glucose ascitic agar to see if any hemoglobinophilic bacilli were present with the meningococci, but there was no evidence of a mixed infection. (3) Twenty cases of influenza meningeal symptoms developed, usually during convalescence. The spinal fluid is different from the meningism occurring in pneumonia, gastro-intestinal conditions, and acute infections in children. It is clear, under pressure, with a slight to a marked increase in the number of cells, with a preponderance of mononuclears, an increase in the albumin and globulin, a normal Fehling's reduction and no organisms.

Johnson [17] reports a case in which the spinal fluid was cloudy and the culture of the fluid showed influenza bacilli and a predominance of polymorphonuclear cells. This occurred in a boy aged 3 years, who had influenza. Convalescent serum was used at three different times with a complete recovery. The so-called encephalitis lethargica undoubtedly belongs in the first group of D'Espine's.[15] A typical case of this puzzling condition is reported by Sharfin.[18] The patient, a 15-months-old infant, died.

Menninger [19] and Barnes [20] describe the mental disturbances and psychoses noted as being postinfluenzal, but make no mention of any children being affected.

Davies [21] reports three cases of nephritis which he thinks are of interest since they all occurred in females under 15 years of age (3, 10 and 14 years), and all cases started on the fourth day of an attack of influenza. He believes that chronic nephritis is a much more common sequel of influenza, but these cases show the importance of examining the urine during such epidemics.

Laessle [22] and Payne [23] had different experiences with the nasal and pharyngeal sequels of influenza. The former took cultures in all

17. Johnson, C. K.: A Case of Influenza Meningitis, Arch. Pediat. **36**:82 (Feb.) 1919.

18. Sharfin, Z.: Encephalitis in an Infant Following Influenza, New York M. J. **109**:576 (April 5) 1919.

19. Menninger, K. A.: Psychoses Associated with Influenza, J. A. M. A. **72**:235 (Jan. 25) 1919.

20. Barnes, F. M.: Psychoses Complicating Influenza, J. Missouri M. A. **16**:115 (April) 1919.

21. Davies, A. L.: Acute Nephritis Following Influenza, Brit. M. J. **1**:73 (Jan. 18) 1919.

22. Laessle, H. A.: Nasal and Pharyngeal Sequels of Influenza, Laryngoscope **29**:103 (Feb.) 1919.

23. Payne, E. M.: Nasal Diphtheria and Epidemic of Influenza, Brit. M. J. **1**:7 (Jan. 4) 1919.

cases that seemed suspicious, with absolutely negative results, while the latter is convinced that the high mortality during the epidemic was owing, in no small measure, to nasal diphtheria which was unrecognized. He reports eight cases occurring among influenza patients in which positive cultures were obtained.

Levin [24] thinks that postinfluenzal alopecia is very common, and reports the case of a girl, aged 14 years, as being typical of many cases seen by him. The alopecia usually appears from three to eight weeks after an attack, and is characterized by a sudden thinning and falling out of the scalp hair, and occasionally by areas of incomplete baldness. The prognosis is usually good.

Of the total number of cases of influenza seen at the South Side Hospital [25] the eye department was called in to see 16 per cent. These were for the greater part cases of acute congestion of the ocular conjunctiva, not a catarrhal type, but marked by a dilatation of the bulbar conjunctival vessels, with some edema of the conjunctiva.

Bronson [26] notes the fact that catarrhal jaundice has always been associated with influenza, yet very few cases have been published in the epidemic. He has notes on twelve children who had a definite exposure to the disease and developed jaundice, but did not develop any signs of the disease itself.

TREATMENT

Prophylaxis.—Classes on the prevention of respiratory diseases [27] have been organized by the department of health of New York City. These classes consist of mothers of enrolled babies and the "Little Mothers," the elder sisters of the station babies. The basis of this work is the instruction given by the department nurses, relating to all the means taken to prevent respiratory infections. Special emphasis is laid on the general cleanliness and ventilation of all the stations, the evils of overcrowding, the avoidance of bringing to the clinic other children than those to be examined, distributing the visits over the entire morning, instead of congregating at one time; group inspection each morning, excluding those with the least sign of respiratory infection, both children and adults, the isolation of children and adults taken ill and the great danger of hand to mouth infection.

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24. Levin, O. L.: Postinfluenza Alopecia, New York M. J. **109**:409 (March 8) 1919.

25. Stieren, E.: Ocular Lesions of Influenza, Am. J. Ophth. **2**:55 (Jan.) 1919.

26. Bronson, E: Catarrhal Jaundice Associated with Influenza in Children, Brit. J. Child. Dis. **16**:78 (April-June) 1919.

27. Weekly Bull. Dept. Health, New York City, Oct. 11, 1919. Preparedness at Baby Health Stations Relative to Influenza.

The use of masks did not meet with much approval. Hill [28] thinks that the use of a mask, by raising the temperature and humidity of the air breathed, is against the natural defensive mechanism, and he urges the deep breathing of cool air brought about by exercise and sleeping in the open air; and as an adjunct, the use of any spray, gargle or snuff, which enhances the outflow of secretion from the respiratory membranes of the nose and throat. Barnett [29] concludes that the wearing of masks is of little value. Their use did not reduce the number of new cases.

Burton [30] had 300 cases of influenza without a complicating pneumonia, and no deaths. He kept them in well ventilated rooms, at an even temperature of 70 F. It is his belief that the influenza bacilli are clinically destroyed or influenced by the temperature to the extent that they will not infect the nurse or persons who may be exposed to the germs of the patient.

La Fetra [7] found nothing specific to combat the disease directly. The vaccines, even for prophylaxis, were not definitely advantageous. The general plan was to have complete rest in bed until the temperature was normal for four or five days. The hot tub, used three or four times a day, was very efficacious. The temperature of the bath should be two or three degrees below the temperature of the patient, and the body should be rubbed thoroughly while the patient is in the tub. For the catarrhal inflammations of the upper respiratory tract, steam inhalations with the addition of creosote proved the most useful. Patients with bronchopneumonia, who have mouth breathing from dyspnea or nasal obstruction, fare much better in a warm, moist atmosphere. After spasmodic contractions are overcome the fresh air treatment is of advantage. For the lobar pneumonias, cold air is of advantage, if there is no nasal obstruction. Digitalis is usually of much help. The course of the disease is very uncertain, and relapses and reinfections of different systems are not infrequent.

Ostheimer,[12] outlining the treatment for infants, lays stress on plenty of fresh air, outdoors on sunny days and the use of magnesium sulphate, from one to four tablespoonfuls, diluted, every morning while the fever lasts. The coal tar products and Dover's powders, reported to be very useful in adults, were not found necessary.

The use of vaccines has been disappointing, although Rosenow,[31] in describing the effects obtained, thought it was possible to obtain a definite degree of protection.

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28. Hill, L.: The Defense of the Respiratory Membrane Against Influenza, Brit. M. J. **1**:238 (March) 1919.

29. Barnett, N.: Masking an Entire School to Prevent the Spread of Influenza, Arch. Pediat. **36**:83 (Feb.) 1919.

30. Burton, S. L.: Uniform Temperature in the Prevention of Influenza and Pneumonia, Southwestern Med. **2**:11 (Feb.) 1919.

31. Rosenow, E. C.: Prophylactic Inoculations Against Respiratory Infections, J. A. M. A. **72**:31 (Jan. 4) 1919.

O'Malley and Hartman [32] report forty-six cases treated by plasma from convalescent patients, and they believe that the toxemia was neutralized this way.

Acidosis, according to Ely,[33] was the chief cause of death during the epidemic. He firmly believes that all patients must be treated for possible acidosis, and to this end advises the use of sodium bicarbonate, potassium citrate and lime water, both at the onset and during the attack.

Prognosis.—This was undoubtedly better in children than in adults, but that it had to be a guarded one is well illustrated by a case reported by Garret.[34] A four-year-old child, who had had influenza two weeks before, had only a slight cough which was improving. One morning, while dressing him, his mother noticed that his body was swollen. At 11 a. m. the trunk, neck and eyelids were swollen and doughy, but no crackling could be elicited. At 12:45 p. m. the swelling had extended to the middle of the forearms and down the thighs almost to the knees. A tympanitic note could be obtained over any part of the swelling, and emphysematous crackling could be elicited on palpation. The swelling of the eyelids was very marked, and the scrotum was blown up to the size of a billiard ball.

At necropsy, pus was found at the bifurcation of the trachea and could be squeezed from the left bronchus. On opening the bronchus, a perforation about one-sixteenth of an inch in diameter was discovered just beyond the bifurcation of the trachea, apparently connected with what seemed to be a suppurating lymph node.

Friend [35] thinks that the extraordinary immunity from influenza among 800 boys at Christ's Hospital school was due, in the main, to the following factors:

1. The physical training, which, although it had been undertaken for only six months, had already produced a marked improvement in physique.

2. The nasal drill, which is perhaps the most important of all. This drill was devised by Ormiston [36] and consists, in the main, of the class room use of the handkerchief and the application of a powder to the nasal membrane to produce free sneezing.

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32. O'Malley, J. J., and Hartman, J. W.: Treatment of Influenza Pneumonia with Plasma of Convalescent Patients, J. A. M. A. **72**:34 (Jan. 4) 1919.

33. Ely, T. C.: The Alkali Treatment Applied to the Acidosis of Epidemic Influenza, New York M. J. **109**:573 (April 5) 1919.

34. Garret, R. R.: Surgical Emphysema Due to Perforation of the Left Bronchus, Brit. M. J. **2**:686 (Dec. 21) 1918.

35. Friend, G. E.: Apparent Immunity from Influenza at a Public School, Lancet **1**:105 (Jan. 18) 1919.

36. Ormiston, I.: Treatment of Adenoids, Lancet **2**:240 (Aug. 24) 1918.

3. The fact that for the past three months the caloric value of the school diet has reached practically the pre-war value of 3,000 calories per boy per diem, for the first time since 1916.

4. The effect of inoculation, both therapeutic and moral. The vaccine used was a polyvalent influenza bacillus vaccine. Mixed vaccines were not used because pneumonia and streptococcus infections were regarded as complications, occurring in the majority of cases after the onset of an influenzal infection.

PNEUMONIA

Cohen [37] and his co-workers have reported some of the results of their studies in pneumonia. They believe that a poison can be demonstrated in the pneumonic lung which is not found in the normal lung. This poison, injected into animals, produces toxic symptoms and prompt death.

Quinin hydrobromid exerted a very marked neutralizing action on the toxicity of the poison isolated. In therapeutic doses it prolonged the life of the animal up to five days after the injection of the lethal dose. Quinin hydrochlorid and quinin, and urea hydrochlorid and ethylhydrocuprein (optochin) afforded less protection. These two points, i. e., the proof of a pneumonic lung poison and its partial conquest by quinin, are new phenomena, and the demonstration is the result of cooperation between the clinician and the laboratory man.

His summary of a definite method of treatment is as follows: Pneumonia patients are benefited by quinin, as shown by clinical and experimental evidence. We can do more good if we give quinin plus digitalis, than if we give digitalis alone, and we can do still more good if we give quinin plus digitalis, plus pituitary extract, than if we leave out the latter. The treatment is not antipyretic, but the temperature is an index to the quinin effect. Pituitary extract, by keeping up the blood pressure, prevents distention, and the indication for pushing digitalis is found in the relationship of diastolic blood pressure to respiration frequency. It is wise to start the digitalis early in order to sensitize the neuromuscular apparatus of the heart.

Alkaline saline drinks or artificial mineral waters should be given freely to keep the urine neutral to litmus.

Behrend [38] calls attention to the necessity of keeping the distinguishing features of the chest versus the abdominal conditions in mind.

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37. Cohen, S. S.: Some Recent Results of Coordination of Laboratory and Clinical Researches in Pneumonia, Pennsylvania M. J. **22**:506 (May) 1919.

38. Behrend, M.: Pneumonia, Acute Empyema, Acute Appendicitis, Pneumococcus Peritonitis; Their Differential Diagnosis, Med. Rec. **95**:102 (Jan. 18) 1919.

Mistakes are made and children are usually the sufferers. Physical signs must assist, but more stress must be laid on inspection, there being only one exception to this observation, when rapid breathing will lead one astray, namely, in that rare condition known as pneumococcus peritonitis. The leukocyte count will be high in both diseases, but it is usually higher in pneumonia. He thinks it would be wise to disregard the blood counts in the differential diagnosis of these diseases.

Lord,[39] as a result of his investigations to determine the influence of varying hydrogen ion concentrations on the proteolytic activity of cellular material obtained from pneumonic lungs, shows that a proteolytic enzyme is present which digests coagulated blood serum at hydrogen ion concentrations of 7.3 to 6.7 and is inactive at higher, i. e., more acid, concentrations. Evidence is also brought forward of a peptone splitting enzyme, operative at hydrogen ion concentrations between 8.0 to 4.8, but most active between 6.3 and 5.2.

The phenomenon of resolution may thus be explained. It is highly probable that during the course of the disease the hydrogen ion concentration of the exudate gradually increases. With the breaking down of the cellular material an enzyme digesting protein (fibrin), in weakly alkaline and weakly acid mediums, may be liberated. With the gradual increase of the hydrogen ion concentration of the pneumonic lung the action of this enzyme probably ceases. An enzyme, capable of splitting peptone to amino-acid nitrogen is probably active during the proteolysis of the fibrin and further activated when the hydrogen ion concentration of the pneumonic lung is increased to within range of its optimum activity, at a p_H of 6.3 and 5.2. By this means it may be conceived that the exudate is dissolved and resolution takes place.

Continuing the study of the relation of the pneumococcus to hydrogen ion concentration, Lord and Nye [40] reach the conclusion that the production of acid is the most important factor in the growth and death of pneumococcus in fluid mediums containing 1 per cent. glucose.

Stadie [41] found no unusually low oxygen capacities while studying the cyanosis of influenza pneumonia, even in the fatal cases, and therefore concludes that methemoglobin formation can hardly have occurred to such an extent as to be a factor.

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39. Lord, F. T.: The Relation of Proteolytic Enzymes in the Pneumonic Lung to Hydrogen Ion Concentrations. An Explanation of Resolution, J. Exper. M. **30**:379 (Oct. 1) 1919.

40. Lord, F. T., and Nye, R. N.: The Relation of the Pneumococcus to Hydrogen Ion Concentration; Acid Death Point and Dissolution of the Organism, J. Exper. M. **30**:389 (Oct. 1) 1919.

41. Stadie, W. C.: The Oxygen of the Arterial and Venous Blood of Pneumonia and Its Relation to Cyanosis, J. Exper. M. **30**:215 (Sept. 1) 1919.

Cecil [42] found pneumococcus vaccine of great value. Among the indications to which he calls attention is the use of it in large institutions, such as orphanages. He believe that the incidence rate of pneumonia would undoubtedly be lowered.

EMPYEMA

Churchill [43] says that this complication is relatively infrequent. The onset is insiduous; early diagnosis is difficult; late diagnosis, with a considerable amount of fluid present, is easy. A displaced apex beat is an important sign. He draws attention to the fact that fluid is found not infrequently in the pleural cavity before crisis, the "pleuresie parapneumonique" of the French. Thus, Jerdine found fluid in 40 per cent. of a small series of cases of pneumonia before the crisis. One to five c.c. of a sterile serofibrinous fluid was obtained on aspiration. This represents the beginning of a possible empyema. Fortunately, few cases "ripen" and the fluid presumably is absorbed. Empyema occurs in about five in one hundred cases, and while this is a very low percentage, the danger signals must be watched for carefully. These are: the child looks sick; is drowsy; begins to have a septic look; the respiratory curve stops its downward convalescent course, remains stationary and then begins to creep up, even before the pulse and temperature have been noted to have changed, the breathing not necessarily being labored.

The conditions likely to be confused with empyema are: unresolved pneumonia; pneumonic abscess and pericardial effusion. Unresolved pneumonia usually gives a dull rather than a flat note; louder and more distinct bronchial respiration; râles, crepitant, subcrepitant and fine moist, and a considerable degree of bronchophony.

Pulmonary abscess is rare in children. Persistent amphoric respiration and a tympanitic quality to the percussion note over a limited area point to this lesion.

Pericardial effusion may simulate a left-sided effusion. The area of dullness, however, is peculiar, being pear-shaped; the heart sounds are muffled and indistinct. A displaced heart beat is an important sign, and in all cases in which fluid is suspected, the exploratory needle should be used repeatedly, if necessary.

Bezy and Escaude [44] present two cases with the hope that it will prevent or help eliminate some of the errors which are made in con-

42. Cecil, R. L.: The Present Status of Pneumococcus Vaccine, Am. J. Pub. Health **9**:589 (Aug.) 1919.

43. Churchill, F. S.: Empyema in Children with Special Reference to Diagnosis, Boston M. & S. J. **181**:87 (July 24) 1919.

44. Bezy, P., and Escaude, F.: Latent Purulent Pleurisy in Young Children, Arch. de méd. d. Enf. **22**:137 (March) 1919.

sidering purulent pleurisy in children. A three-year-old child had had at eighteen months what had been disgnosed bronchitis and pertussis. The child was never well after that, and was considered to have a tracheobronchial adenopathy. Months later a swelling appeared in the axilla which ruptured spontaneously and discharged pus at intervals. The case was then diagnosed as tuberculosis of one of the ribs, but a fuller consideration of the case, helped out by the roentgen ray, led to a diagnosis of purulent pleurisy. Resection of the rib was followed by a quick and complete recovery.

The second case was in a two-year-old boy who gave physical signs of empyema, but in whom two exploratory punctures were negative. A tumor-like swelling appeared later in the left mammary region, puncture of which proved the presence of pus, which contained pneumococci. Rib resection resulted in a cure.

Special emphasis is laid on the necessity of repeated roentgen-ray examinations of all difficult, long-standing chest cases.

The treatment of empyema has been the subject of many papers lately. The report of the United States Army Empyema Commission leans more to the wider employment of the Carrel-Dakin method than to any other single method, but as yet, reports of its use in children are very meager. Brooks [45] reviews the newer methods of treatment, including the repeated aspirations following with injections of neutral solution of chlorinated soda, a closed method used at Walter Reed Hospital and the Carrel-Dakin treatment as carried out at the Rockefeller Institute.

Lilienthal [46] thinks that in deciding on the primary operative procedure in any case of empyema, the first thing to consider is whether or not it is the mechanical pressure of the purulent fluid on the circulatory apparatus in the thorax, with the displacement of the heart, which is causing the cyanosis and respiratory embarrassment so frequently noted. The loss of respiratory area is not enough in itself to account for these symptoms, nor is the sepsis. Relief of pressure is the first crying need and must be secured as soon as possible. If the patient is acutely ill and in great distress with seropurulent exudate, the thorax can easily be emptied without shock by what is known as air replacement aspiration. This, in fact, is not at all an aspiration in the true sense of the word, but a mere evacuation of the fluid by means of a small trochar and cannula which permits the fluid to escape and a certain amount of air to replace it without the powerful vacuum force of the usual Potain or other suction apparatus.

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45. Brooks, C. D.: Treatment of Empyema, J. Mich. M. S. **18**:107 (March) 1919.

46. Lilienthal, H.: The Selections of Operative Methods in Empyema of the Thorax, New York State J. M. **19**:359 (Oct.) 1919.

In commenting, in his summary, on the Carrel-Dakin method he says that the method may be employed with excellent results when the cavity is simple and the lung expansile. It is not to be used in complicated cavities when the tubes cannot be made to reach every part.

Emerson [47] claims that among certain points of great importance in the surgical care of empyema, one that is still not clearly recognized is that no operation should be performed early. So long as the quantity of pus is not so large that by its mere bulk it interferes with the action of the heart and lungs, there is no great hurry in removing it.

Chevrier [48] believes that the logical treatment of purulent pleurisy is the use of a disinfecting gas or vapor combined with a double thoracotomy. He lays much stress on the necessity of having the patient in the proper position, claiming that in the empyema operations as usually performed not enough attention is paid to the proper emptying of the costophrenic sinus nor the costovertebral groove. His proposed technic is described in detail and well illustrated.

Thomas [49] describes the essential features of an encapsulated empyema rupturing into the pleural cavity. It usually occurs during the course of a normal convalescence, being ushered in with a sharp pain in some part of the chest, brought on by slight exertion. In a few hours, the patient becomes critically ill with a rise in temperature, rapid pulse and increasing dyspnea. The abdomen is found to be greatly distended and a generalized tenderness is present. The patient becomes progressively worse with no change in the picture, except an increasing prominence of the signs of fluid in the chest.

BRONCHITIS

Patterson [50] considers it timely, in view of the transference of the peanut from a delicacy into a more staple article of diet, to draw attention to and consider the seriousness of the peanut as a foreign body in the bronchi. Six cases are reported, all in children less than two years of age. Attention is drawn to the fact that septic pneumonia often follows in this class of foreign bodies; that small fragments of peanuts at times are held in the swollen mucosa, and that they are not coughed up. Often in cases where the foreign body is opaque to the roentgen ray the physical signs are the sole reliance; but too often the signs are considered indicative of the lesions present, but the foreign

47. Emerson, C.: Treatment of Empyema by the Closed Method, Nebraska M. J. **4**:65 (March) 1919.

48. Chevrier, L.: Study on the Drainage of the Pleura, Presse méd. **27**:9 (Jan. 9) 1919.

49. Thomas, H. M.: Rupturing Encapsulated Empyema Into the Pleural Cavity, J. A. M. A. **72**:29 (Jan. 4) 1919.

50. Patterson, E. J.: Peanut Bronchitis, New York M. J. **109**:101 (Jan. 18) 1919.

body origin is unsuspected. Occasionally, after bronchoscopy, tracheotomy is necessary to prevent a child dying in his own secretions. Bowen [51] stresses the same point. For a variety of reasons, unsuspected foreign bodies in the lungs are of frequent occurrence in children, much more frequent than previous experience has indicated. The usual diagnosis is chronic bronchitis, less frequently it is slowly advancing tuberculosis. The average general practitioner is far too apt to discount the probability of the presence of a foreign body in the lung.

FETAL BRONCHIECTASIS

Koecker [52] reviews the literature of the two types of congenital bronchiectasis and describes an interesting type of the fetal kind, discussing some points suggested by his own case. The cause of fetal bronchiectasis is unknown, but its bronchial origin cannot be doubted. Among modern investigators the current belief is that it is a primary maldevelopment.

ASTHMA

Scheppegrell [53] states that 5 per cent. of hay-fever cases develop before the age of 5 years, and 24 per cent. before the age of 24 years. It is relatively common among children, but it is not recognized as the diagnosis is usually "a cold."

The initial sensitization of hay-fever pollen in children is usually due to the direct inhalation of certain wild flowers. A common one is the daisy fleabane, *Erigeron strigosus,* found usually in neglected fields and on roadsides. It has some resemblance to the common daisy, and is therefore frequently collected by children. Another is the common dandelion, *Leontodon taraxacum.* The pollen is quite noxious and often causes an attack of hay-fever which is usually mistaken for a cold. This is not listed on the hay-fever weed list because it is not wind pollinated.

The field daisy, black eye susan, and others of the "compositae" family are insect pollinated and thus do not infect the atmosphere for hay-fever sufferers, but if applied directly to the nostrils, and the pollen is inhaled, an anaphylaxis may develop, resulting in a sensitization to other members of the same group. As ragweed is a "compositae," the sensitization may result in a persistent fall hay-fever.

Initial attacks are usually due to increased exposure; these are followed by an anaphylaxis to atmospheric pollens which previously failed to produce an attack.

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51. Bowen, D. R.: The Unsuspected Foreign Body as a Frequent Cause of Chronic Bronchitis, Am. J. Roentgenol. **6**:111 (March) 1919.

52. Koeckert, H. L.: Fetal Bronchiectasis, Am. J. Dis. Child. **17**:95 (Feb.) 1919.

53. Scheppegrell, W.: Hayfever in Children, Med. Rec. **96**:97 (July 19) 1919.

As in adults, asthma is sometimes the predominant symptom of hay-fever in children. When asthma is marked during the hay-fever season, the reaction to pollens is probable. The treatment is outlined and the results of this have been very encouraging.

Donelly,[54] discussing the etiology, symptomatology and treatment of bronchial asthma in children, calls attention to the fact that in the sensitization tests there is a marked reaction, in a large percentage of cases, to the protein of walnut, and he believes that not enough attention has been given to this point. Walker's [55] conclusions, following his studies on the treatment of asthma with vaccines, are as follows: Twenty-eight patients with bronchial asthma were treated with vaccines of the bacteria to which they were sensitive. Seventy-five per cent. were relieved of asthma and 21 per cent. were improved. Seventy-five patients were treated with vaccines made from culturing their sputum on plain agar. The predominating organism was usually the one selected for treatment. Forty-six per cent. were relieved of asthma and 16 per cent. were improved. These were all nonsensitive patients. Twenty-four patients were treated with vaccines made from culturing their sputum on dextrose bouillon and using only the streptococci. Thirty-seven per cent. were relieved of asthma and 25 per cent. were improved. These were also nonsensitive patients. Sixteen nonsensitive summer asthmatics were treated with vaccine. Thirty-one per cent. were relieved and 25 per cent. were improved. Thirty-five nonsensitive patients were treated by vaccines made by culturing their sputum both ways; in other words, many types of vaccines were used. Thirty-one per cent. were relieved and 23 per cent. were improved. With the sensitive cases, the age of onset of asthma, the duration of the asthma and the age of the patient when treated, had little to do with the prognosis. However, with the nonsensitive cases these facts had much bearing on the prognosis. The older a patient is when an asthma begins, and the older he is when treatment is begun, the more unfavorable the prognosis from vaccines in nonsensitive cases. Therefore, in general, the younger a patient is when asthma begins the better the prognosis, and in children the prognosis is good.

Walker [56] states further that patients who have seasonal bronchial asthma caused by pollens are prevented from having asthma by a series of treatments with the pollens to which they are sensitive, provided sufficient treatment is given. Treatment with pollens during the season is less reliable.

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54. Donnelly, W. H.: Bronchial Asthma in Children, New York M. J. **109**: 503 (March 22) 1919.

55. Walker, I. C.: The Treatment of Bronchial Asthma with Vaccines, Arch. Int. Med. **23**:220 (Feb.) 1919.

56. Walker, I. C.: Sensitization and Treatment of Asthmatics with Pollens, Am. J. M. Sc. **157**:409 (March) 1919.

The intestinal tract is considered by Lewis [57] the "fons et origio mali." Poisons produced there are carried through the body, react on the nervous system in some way and produce the asthmatic attack. The treatment is to modify the diet so as to prevent the formation of poisons.

Brown [58] divides asthmatics into two groups — the first having definite pulmonary signs, and the second, in which there is nothing more than a transient obstacle to respiration, with little or no secretion in the bronchial tree. The first group is further divided into two subgroups, one in which no nasal deformity or disease of the accessory sinuses exists, and a second in which one or both are present.

The original second group is likewise further divided into three subdivisions — the first, in which the nervous element is a causative factor, the second he refers to as a nasal reflex asthma, and a third in which there is no irritable nasal condition present, but there is a definite respiratory circulatory reflex.

Lorie [59] operated in thirty-nine cases of asthma and obtained thirty-five cures. He believes in the most thorough examination of the nasal passages of all cases of asthma. The case of a twelve-year-old girl is cited, who had asthma since childhood. The tonsils and adenoids were removed without any improvement. Examination finally revealed a nonsuppurating ethmoiditis. Operation resulted in a complete cure.

Sawtelle,[60] on the other hand, says that the extravagant claims for the cure of bronchial asthma by intranasal surgery alone are unsupported by scientific investigation and careful clinical observation.

RETROPHARYNGEAL ABSCESS

Ninety-six per cent. of these abscesses begin before the sixth year, and 50 per cent. occur in children between the ages of 6 months and 1 year, according to Brown.[61] The diagnosis presents some difficulties to the general practitioner, as only one of the cases reported was correctly diagnosed.

These patients usually have some history of a nasopharyngeal infection, or some illness which can be traced to an infection of the upper respiratory tract. The primary condition may have been entirely recovered from or may have merged into the symptoms presented by

57. Lewis, P.: Asthma, Med. Press **107**:160, 1919.

58. Brown, R. G.: Asthma from the Point of View of the Rhinologist, M. J. Australia **2**:463 (Dec. 7) 1918.

59. Lorie, A. J.: Nasal Sinuses and Asthma, J. Missouri M. A. **16**:113 (April) 1919.

60. Sawtelle, J. E.: The Relation of Focal Infection and Protein Poisoning to the Upper Respiratory Tract, J. Kansas M. S. **19**:25 (Feb.) 1919.

61. Brown, J. M.: Acute Retropharyngeal Abscesses in Children, Laryngoscope **29**:9 (Jan.) 1919.

the retropharyngeal abscess. As the abscess develops, there is a return of the fever or an increase in that which was already present, together with general symptoms that accompany any infectious process.

The local symptoms are some interference with respiration and deglutition. Because of this, many cases are diagnosed croup. The dyspnea is usually inspiratory in type, this being one of the best diagnostic symptoms. Choking spells are common; the child almost strangles, and in one of the cases reported tracheotomy had to be done to restore breathing. Any child having trouble with respiration and deglutition should be regarded as a possible subject of a retropharyngeal abscess, and, if necessary, a digital examination of the pharynx should be made.

He regards it as a very serious lesion if undiagnosed, and does not believe in allowing spontaneous rupture. One of the patients whose case is reported undoubtedly choked to death following a spontaneous rupture, the small larynx having been filled with pus during a sudden inspiration.

The only rational treatment, Marfan [62] says, is operation, and this should be done immediately. The inside method is much preferred.

ROENTGEN RAY IN THE DETECTION OF FOREIGN BODIES

Jackson [63] has an excellent résumé of cases of foreign bodies in the air passages, presenting particularly the pathology of different types. Bowen [64] reporting the case of a boy, aged 8 years, who had had a foreign body in his lungs for five years, pleads for the use of the roentgen ray in routine examination of the thorax, if for no other reason than to eliminate the question of foreign body.

PHYSIOLOGY

Meakins [64] applies lessons learned while investigating respiratory disturbances due to gas irritation, to those occurring in civil practice. Pneumonia dyspnea, he believes, is due to an oxemia primarily, and the usual method of treating the dyspnea, associated with cyanosis and vasomotor paralysis, with oxygen is all wrong. The oxygen must be used intelligently, and the proper way is that devised by Haldane, involving the use of a mask.

The significance of Hoover's [65] observations lies in the recognition of disassociation between anoxemia and carbon dioxid of the blood.

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62. Marfan: Acute Retropharyngeal Abscess, Med. Press **1**:468, 1918.
63. Jackson, C.: Observations on the Pathology of Foreign Bodies in the Air and Food Passages, Surg., Gyn. & Obst. **28**:201 (March) 1919.
64. Meakins, J. C.: Causes and Consequences of Disturbances of Respiratory Rate and Rhythm, Canadian M. A. J. **9**:319 (April) 1919.
65. Hoover, C. F.: Moisture in the Air Spaces of the Lungs and Oxygen Therapy, Tr. Sect. Pharmacol. & Therap., A. M. A., p. 103, 1918.

Unless the entire volume of blood passing through the pulmonary circulation is exposed to a respiratory membrane which has a uniform exposure to the ventilating air of the lungs, the analyses of expired air cannot be employed to interpret the oxygen and carbon dioxid content of the blood.

MISCELLANEOUS

Bennett [66] insists that the purity and not the coldness of the air is the sine qua non of respiratory treatment. He believes that many of the recently published opinions against the use of alcohol in pneumonia are brought about, partly, at least, by the present wave of prohibition. Coal tar products are tabu, and pneumonia patients are rarely examined, once the diagnosis is made.

Analyzing some of the facts relating to the frequency of acidosis in children, Stevens [67] believes that infection of the upper respiratory passages may be an etiologic factor. A preliminary survey of several cases, treated with that viewpoint in mind, were presented.

Osborne [68] calls attention to the fact that colds, especially in children, are to be considered regrettable occurrences and should always be treated properly and never neglected. He suggests the use of $\frac{1}{500}$ grain atropin for a child 10 years old, only, however, in the early stages of the disease. This is to be given every three hours, for five doses, then every six hour, for five more doses. This is a valuable adjunct in addition to other methods which he describes.

An excellent discussion of ventilation is given by Reed [69] with a good bibliography. In his summary he states that because the effects of fatigue are now recognized as being similar to those caused by poor ventilation, it is apparent that there is a close relationship between the two conditions, and it may be that the effects are the same though the mechanism is not clearly outlined.

Pulmonary syphilis [70] is seldom diagnosed until necropsy. The chief forms are: gumma, bronchiectasis and pneumonia.

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66. Bennett, C. D.: The Cult of Cold, J. M. Soc. New Jersey **16**:84 (March) 1919.

67. Stevens, F. A.: Acidosis and Its Relations to Upper Respiratory Conditions, J. Iowa S. M. S. **8**:136 (April) 1919.

68. Osborne, O. T.: The Etiology and Treatment of Colds, New York M. J. **109**:529 (March 29) 1919.

69. Reed, C. I.: Why Is Poor Ventilation Harmful? Am. J. Pub. Health **9**:589 (Aug.) 1919.

70. Canelli, A. F.: Congenital Pulmonary Syphilis, La Pediatria **27**:11 (Jan.) 1919.